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XLVIII.

TUBERCULOSIS OF THE MOUTH.*

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Tuberculous lesions of the mouth include those involving the lips, cheeks, gums, hard palate, soft palate, tongue, teeth and alveolar process. Tuberculosis of the pharynx is not included in this paper, although it is difficult to draw the dividing line. So large a subject as tuberculosis of the tonsils forms a subdivision of pharyngeal tuberculosis and is also omitted from this discussion, although it will be necessary for purposes of comparison and illustration to touch upon all lesions of tubercular character ordinarily seen by what is known as pharyngoscopy. It is comparatively rare to see isolated lesions involving but one of the structures included in the mouth, and it is the author's belief that such rare cases form a class by themselves, possessing the same general

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characteristics that other tubercular lesions of the upper air passages do, but marking a distinct difference in course and prognosis.

A careful review of over 200 references, abstracts of which will appear in a subsequent paper, impresses one with the rather large number of tuberculous lesions of the mouth which have, from the time of Thomas' article[1], 1839, been reported. The largest number, however, have appeared within comparatively recent years, and I wish to mention especially the early articles of Bosworth[2], DeBlois[3], Delavan[4]. Yet when one analyzes the extensive literature, he must be struck by the general inaccuracy in diagnosis and the lack of appreciation of distinct variations and differences in these lesions, differences which are of special importance in etiology and prognosis.

Forms.—Tuberculous lesions may manifest themselves in different forms, according to pathologic changes, according to mode of development and according to their clinical course.

First, the various pathologic alterations seen are nodular infiltration, superficial ulceration, deep ulceration, perforating ulceration, necrosis of bone, chronic abscess and tumor.

Second, according to the mode of development, we have two forms, "endogenous," and "ectogenous" (Grunwald[5]), which corresponds to the general classification of Hollander[6] into "descending" and "ascending."

Third, according to clinical course, we may have "malignant" and "benign."

Clinical observation confirms this classification. The "ectogenous" or "ascending" form, that which may be designated as the inoculation variety or purely local, represents the less active, sluggish or "benign" type, while the "endogenous" or "descending" variety, that which represents infection through blood and lymph streams, through miliary deposits or infection from within, corresponds to the more active, virulent, "malignant" type.

Etiology.—It is a well established fact that tuberculosis of the upper air passages, and particularly of the mouth and pharynx, is more commonly found in males than females. This leads to the question of how far local irritation of the mouth and possibly slight injury of the mucous membrane enter into the causation of tuberculosis. This also raises the question as to the existence of primary tuberculosis of these structures. There are innumerable cases upon record in which

the only demonstrable lesion is that localized in the mouth and especially upon the tongue. In these cases, the history of ulceration following a slight injury to the mucous membrane, which although nothing more than an abrasion to begin with, refused to heal, should be fairly conclusive proof that the abraded surface was an easy portal for infection in which tubercle bacilli became lodged, the characteristic tubercular process following. On the other hand, assuming that there is a necessary vulnerability without which tubercle bacilli may be innocuous, leads to the belief that there already exists in individuals developing such a localized tuberculosis, another and primary focus of infection. Auguy[7], 1895, and others since then have stated that the most frequent mode of infection is through the blood current. Walsham[8] has shown the importance of the lymphatic system in the development of tuberculosis, especially with regard to the frequency with which infection may be conveyed to the bronchial glands from above, through the adenoid tissue in the throat, including the pharyngeal and faucial tonsils. It has been unquestionably demonstrated that tuberculous infection may invade the system through the tonsils without producing any alteration in the tonsils themselves. It has also been shown by Cook[9] that the teeth, especially when diseased, form excellent foci through which infection may be carried to the adjacent lymphatic structures. The question, therefore, becomes an extremely complicated one, especially when we remember that post-mortem examinations have revealed the presence of foci of tuberculosis in the lungs which were not suspected and could not be demonstrated ante mortem. It seems reasonable to conclude that although local or systemic causes exist, infection may descend through the lymphatic system to the bronchial glands, invade surrounding structures and be carried by the blood current to such parts as the tongue, the lips, the gums or the hard palate, and that additional local irritation or trauma, however slight, may determine the outward manifestation of tuberculosis in these regions. Ragged, sharp or decayed teeth, extraction of teeth, poorly fitting dentures, biting the tongue, injury from pins or other sharp bodies in the mouth constitute the more common local causes. In fact when we consider that the mucous membrane of the mouth, although more or less constantly bathed in sputum laden with tubercle bacilli in tuberculous individuals, develops tuberculous lesions

but rarely, one must admit that there exists a special protective agency. This protective agency obtains so long as the surface of the mucous membrane is not altered either by traumatism or pathologic changes. Once this protective agency is in a measure diminished, local tuberculosis may manifest itself. The more frequently one sees tuberculosis of a given organ, the more reasonable it is to presume that the infection of that structure came about through the blood or the lymph current. The less frequently certain structures are involved, the more reasonable to suppose that some local cause must operate to overcome its invulnerability. This thought is supported in the classification of Grunwald, Walsham and others, as well as in the clinical history. It is a recognized fact that the socalled primary lesions of the mouth are more sluggish and less malignant than the secondary varieties, the latter developing through blood and lymph current in an organism abundantly attacked by the tuberculous process. This thought is further borne out by the slow and comparatively non-malignant affections of the gums, the hard palate, the lips, the cheeks and the tongue, and the rapidly progressing, actively malignant course of tuberculosis of the pharynx and tonsils.

Subjective Symptoms.—Tuberculous lesions involving the tongue, the lips, the gums, may exist for some time before the patient is aware of their presence. Even ulcerating lesions may develop to a considerable extent before attracting attention. In this respect these lesions differ from those involving the pharynx and larynx. The earliest manifestation is a sense of slight soreness or burning, marked particularly when the diseased surface is irritated by contact with food. Soreness may become decided pain, more especially when the lesions involve the tongue. When seen upon the hard palate or gums the pain rarely becomes severe, differing from the usual intense pain of tuberculous ulcers. There is localized swelling and slight increase in the secretion of the parts, which becomes viscid, grayish white or dirty. A slight odor is often apparent although rarely becoming offensive. Even when the ulceration has gone on to marked necrosis of the underlying structures, the odor that is at times discernible may be attributed to the general cachexia of the individual rather than to the local lesions. Glandular involvement may or may not be present. I have seen it in very early lesions and have failed to discover any enlargement of cervical glands in cases far

advanced. When it does exist it is rarely painful. In tuberculosis of the tongue, lips, gums or palate, the general symptoms of the patient are comparatively slight except in those cases which develop in the course of severe general infection, such as is found in a miliary process. These severe cases are usually marked by the rapid development of lesions extending to the soft palate, the tonsils and the surrounding pharyngeal structures, when dysphagia and odynphagia of severe type, high temperature, rapid pulse, marked emaciation and exhaustion may be added to the symptoms.

Objective Symptoms.—Excluding those isolated instances of tumor and abscess[10], there is apparent to the most casual observer a definitely uniform local lesion. The general appearance is that of a pale, superficial ulceration without inflammatory areola, edges irregular in outline and beveled or undermined, tending to spread laterally, not deeply. A viscid, dirty white secretion covers the ulceration, which when cleaned away brings to view a more or less nodular appearance. Scattered irregularly over the surface of the ulceration and upon its margins are seen small, red, soft granulations, interspersed with *pin-head spots of yellow or gray.* These yellow spots, the spots of Trelat[11], may be seen also upon the mucous membrane adjacent to the ulceration.

Case I, Figures 1 and 2, demonstrates very early as well as moderately advanced tuberculosis of the gums. This patient, aged 26, was first seen in December, 1906, having been referred by Dr. Bergtold. He had had pulmonary tuberculosis one year. In August, 1906, the gums became sore. There was also some soreness in the nose and larynx, the latter inducing painful and difficult swallowing. A typical tuberculous ulcer was seen upon the left surface of the nasal septum. The larynx presented pale infiltration of both arytenoids with numerous small ulcers and grayish deposits. The gums over the first bicuspid on the right side of the upper jaw and over the first and second bicuspids right side, lower jaw, were seen to be covered with small reddish nodules, very superficial ulcers and a few pin head yellowish spots. The typical tuberculous appearance of a very early lesion was readily demonstrated. Fig. 1. Upon the lingual surface of the gums of the first and second molars, lower jaw, left side, a more advanced lesion was seen, possessing, however, the same characteristic nodular superficial ulceration with reddish and yellowish spots.

Fig. 2. Although typical in appearance, repeated and thorough examination of curettings of these lesions by Dr. Todd failed to demonstrate the presence of tubercle bacilli.

The margins of the ulcerations are rarely indurated. Upon the tongue, however, a slight induration may be felt and especially where healing has occurred, leaving the organ markedly fissured. The character of these indurations is a fibrosis, which has occurred in the healing process.

Case II, Figures 3 and 4, shows a typical case of tuberculosis of the tongue. Figure 3 illustrates the fissured appearance which has resulted in the healing process, the induration on the margin of these fissures being marked. Figure 4 shows an active lesion at the tip of the tongue, presenting all the usual characteristics of tuberculous ulceration. This case was that of a man aged 35, who had had tuberculosis for seven years. He came to Colorado immediately upon development of the trouble. Eleven years ago, or four years before pulmonary tuberculosis was detected, he had a small superficial ulcer upon the dorsum of the tongue, which refused to heal. Subsequently, similar ulcerations on the dorsum developed and continued active for an indefinite period. The patient cannot state exactly when healing began. One year ago the ulceration upon the tip developed, which was extremely painful, a symptom which did not obtain in the ulceration upon the dorsum. When seen at the National Jewish Hospital for Consumptives in December, 1906, the healing process upon the dorsum of the tongue was complete with the exception of two small lesions. The tip of the tongue, however, was in a condition of active ulceration. Careful curettings from the dorsal as well as tip ulcers, showed tubercle bacilli in intimate relation with the epithelial cells. At the present time, May 23, 1907, the patient's general condition is slowly failing, the ulceration upon the tongue, both dorsal and tip, are perfectly cicatrized.

When the disease extends to the soft palate, the anterior or posterior pillars, the uvula or the tonsils, a somewhat different picture presents itself. The very earliest appearance is a marked pallor which is heightened by an edematous swelling. This edema may be extremely slight but it gives to the structure a characteristic appearance. Very careful observation will reveal the presence of numerous yellow or grayish pin head spots just under the surface of these pale, edematous structures.

This condition is typically illustrated by the following cases: Case III, Fig. 5. C. Mc., male, aged 28, referred to me October 2, 1906, by Dr. S. Solis-Cohen of Philadelphia. The patient had slight pulmonary involvement and a tuberculous ulcer of the left arytenoid and vocal band. These lesions had improved under Dr. Cohen's treatment, the improvement continuing after coming to Colorado. On the 27th of February, 1907, both tonsils were swollen, pale and dotted with a few small, grayish deposits. The posterior pillars were slightly edematous. The patient's general condition was failing. A small piece of tonsillar tissue was removed and sections made by Dr. Todd, who reported numerous miliary tubercles with giant cells and caseous centers. A few tubercle bacilli were demonstrated at the edges of the caseous areas.

Case IV, Figure 6. Male, aged 24, referred by Dr. Levitt of New York. This patient was presented before the section of Laryngology at the New York Academy of Medicine by Dr. Emil Mayer, February 27, 1907, demonstrating a possible primary lupus of the larynx. Upon examining the patient March 9th, there were found slight pulmonary involvement, swollen epiglottis, which was red and covered with pin head grayish tubercles and notched in the center, probably from a section removed. The aryepiglottic folds were swollen, pale, edematous and covered with grayish deposits. The arytenoids were moderately swollen and pale. The patient's condition rapidly grew worse, and upon April 9th there was discovered on the right tonsil a whitish deposit. The left tonsil presented a small, irregular, nodular ulceration with whitish imbedded masses. Scrapings from both tonsils were submitted to Dr. Todd, who reported as follows: The cover glass smears from the left tonsil show numerous cells, the structure of which cannot be clearly seen. Careful search shows very few tubercle bacilli scattered among them. The cover glass preparation from the right tonsil contains many squamous epithelial cells and a few other cells which cannot be made out distinctly, and some degenerated material. No tubercle bacilli can be found. Figure 6 shows distinct difference between the two tonsils, the right presenting an exudate upon its surface while the left shows the typical tubercular appearance. The difference in the two tonsils as demonstrated clinically is borne out by the laboratory report.

Following the deposit of tubercles, ulceration rapidly de-

velops, the yellowish spots breaking down and coalescing in an irregular manner, giving to the surface of the ulceration the characteristic worm-eaten or mouse-nibbled appearance. When the ulcerations become deep, as they sometimes do, they still retain upon their margins the characteristic tubercular appearance. This is demonstrated by the following case:

Case V, Figure 7. Male, aged 44, advanced tuberculosis of the lungs and larynx of two years' standing. Three weeks ago the gums became sore and an ulcer developed upon the upper jaw. This seemed to be the result of loose and decayed teeth, which were plainly apparent. The ulceration is a typical deep tubercular ulceration extending to the alveolar process, which is itself necrosed, showing an exposed tooth root. A section of the margin of the ulcer was removed and the laboratory report gives the following: Granulation tissue and a few well defined tubercles with caseous centers; small number of tubercle bacilli scattered about the periphery of the tubercles.

Extensive ulcerations involving the soft palate and posterior structures are illustrated by:

Case VI, Figure 8. Male, aged 40, advanced tuberculosis of the lungs, sore throat at times during the past seven years. For three weeks past there has been painful and difficult swallowing. Examination of the pharynx shows extensive mouse-eaten appearance involving soft palate, both pillars of the fauces, tonsils, uvula and posterior wall of the pharynx downwards to the left side of the larynx. The ulcerations extend forward as far as the junction of the soft with the hard palate.

Diagnosis—The lack of scientific accuracy so frequently displayed in the diagnosis of tuberculosis of the mouth and pharynx is worthy of note. Any or every ulceration of the mucous membrane of the mouth or pharynx in a tuberculous individual is not necessarily tuberculosis. It is feared that this has too often been believed and that it may be the reason so many cases of cure have been reported.

Illustrating this I present:

Case VII, Figure 9. H. T., aged 32, tuberculosis of the lungs, one year. Ulceration on the under surface of the tip of the tongue for six months. These ulcerations are two in number and are not painful. They present a marked red, inflammatory areola, are irregular in outline and show nowhere any of the characteristic grayish or yellow spots, or reddish granulations. They are evidently due to contact with the

sharp lower incisors, aggravated probably by more or less persistent coughing. When last seen, May 19th, the lower ulceration had entirely healed without treatment.

The diagnosis is certain when there is seen a *pale ulceration, without* inflammatory areola, *superficial, worm-eaten* in appearance, dotted with reddish pin head elevations and having *small yellow* or *grayish spots* scattered throughout. An early diagnosis when the soft palate or tonsils are involved may be assumed when there appears an extremely *pale, slightly edematous mucous membrane* with *small submucous, pin head yellow spots.*

These diagnostic features, however, are not always present, although they do exist in the vast majority of instances. In all cases the diagnosis should be confirmed by the microscopic findings, and in obscure cases this and the inoculation of guinea pigs are the only positive tests. The finding of giant cells and caseous material together in sections is almost certain proof of tuberculosis.

In case of the ulcers, the laboratory confirms the diagnosis, according to Dr. J. C. Todd of the Pathological Laboratory of the Denver and Gross College of Medicine, in two ways:

1. Detection of tubercle bacilli, (a) by the microscope, or (b) by inoculation of guinea pigs.

2. Detection of the histologic structures of tubercle in stained sections.

(1) Detection of the bacilli:

(a) Owing to possible presence of tubercle bacilli in the mouth, simple examination of swabs from the surface of the ulcer is of little value. The surface of the ulcer should be thoroughly cleansed and then curetted under cocain; or a piece of sufficient size for sections should be excised. Curettings are to be thoroughly rubbed between slides or cover glasses until the cells are sufficiently dissociated to allow thin smears. The presence of tubercle bacilli in the smears may be taken as proof of tuberculosis, provided the surface of the ulcer was well cleansed, and this is made absolutely sure by finding the bacilli within the small clumps of cells which the rubbing between slides failed to separate.

When a piece of sufficient size can be excised, it should be sectioned and stained for tubercle bacilli. Tubercle bacilli are sometimes abundant even when the structure of the tubercle is very doubtful.

(b) Inoculation of guinea pigs is resorted to only when other means fail.

(2) The histologic structure of the miliary tubercle can generally be found in portions of tissue which have been sectioned and stained. Recognition of the tubercle depends upon the presence and, especially, the arrangement of certain structures; no one structure is diagnostic in itself, although its presence may be very suggestive. However, the presence of giant cells and caseation *together* may generally be accepted as proof when the structure is not otherwise typical.

Grunwald[12] has shown how difficult it is to find tubercle bacilli in all tuberculous ulcerations. Hajek[13] has also called attention to this fact in tuberculosis of the nose, therefore, one can not place all of his reliance upon the presence of these organisms. With few exceptions will one fail to find them present if sufficiently long and accurate search be made. They are usually few in number and scattered and are rarely absent in typical lesions. In the case presented under Figures 1 and 2, the local lesions were absolutely typical in appearance, but in spite of most thorough and repeated search, tubercle bacilli were not found.

Prognosis.—When the affection involves the structures anterior to the soft palate, including the tongue, gums, lips, cheek, hard palate, the prognosis so far as the general lesion is concerned is of comparatively little importance.

Case VIII, Figures 10 and 11. V. G. L., aged 58, has been ill with tuberculosis of the lungs for over fifteen years. Slight ulceration of the hard palate and gums were noticed in October, 1906. These lesions have been comparatively free from pain. There is, however, marked soreness and discomfort, especially upon eating. Figure 10 shows a typical and characteristic tuberculous ulceration of the hard palate, with beveled edges and nodular base. Figure 11 shows more advanced tuberculosis of the gums, rather deeper than usual, with undermined edges, especially inferiorly. Careful curettings from both of these ulcers were examined by Dr. Todd, who easily demonstrated tubercle bacilli, the entire absence of all other bacteria showed the thoroughness with which the parts were cleansed and proved conclusively that the tubercle bacilli were obtained from the ulcerations themselves and were not the result of contamination.

Tuberculous ulcerations rarely heal, but at the same time

their progress is extremely slow. They are the source of but little discomfort and have no special bearing upon the course of an associated general or pulmonary tuberculosis except in so far as they indicate involvement of a small amount of additional tissue. Some of these cases have been said to heal spontaneously, others to have been cured by excision or cauterization. Unquestionably a few rare instances of such cures may be accepted as authentic, such cases representing that comparatively benign form described as "ectogenous" or "ascending." Bernheim[14] states that in bucco-pharyngeal tuberculosis a cure is the rule. Gleason[15] also states the "Prognosis as regards healing is favorable." So firmly convinced am I that these statements are erroneous, that I venture to question the diagnosis.

When tuberculous lesions involve the soft palate, uvula, the tonsils, anterior pillars and the structures posteriorly, the prognosis is of very much more significance. Not only is the prognosis of the local lesions absolutely unfavorable, but their effect upon the general condition of the patient is so deleterious that one can predict a rapid decline and an early and fatal termination. Lesions here are positive evidence of rapidly disseminating miliary tuberculosis.

Two exceptions to the above statements should be made. First, when the lesion involves the tongue the prognosis may be quite as unfavorable as when it involves the pharyngeal structures, and, second, comparatively benign lesion may exist in the posterior wall of the pharynx.

Treatment.—This consists in palliative and curative, and should be both local and general. Palliative treatment is directed to the relief of pain. Cocain or powdered orthoform applied locally are the most valuable remedies. Pain may also be mitigated by curetting and cauterizing, on the theory that the pain is due not so much to the exposed nerves as to the development of small neuromata upon the exposed nerve ends.

Curative.—Ulcers which involve a small portion of the tip or margin of the tongue, or localized tumors have been removed by more or less extensive radical excisions. Temporary healing may be brought about by thorough cauterization, followed by frictional rubbings with lactic acid or formalin. Galvanocautery has been of some avail and particularly have I seen early tubercular ulcers of the tonsils, lips and tongue heal

under this treatment. Usually these healed ulcers reappear or there develop others adjacent to them. In spite of the generally accepted ultimate fatal outcome of these cases, one should not neglect an attempt at cure, and therefore, in addition to local treatment, the usual attention should be paid to the patient's general welfare. Rest is of prime consideration, owing to the frequent presence of high temperature. The patient's nutrition should be maintained at the highest possible standard by the administration of large quantities of easily digested, nutritious food. When much pain exists this becomes a question of serious moment, and local anesthetics should be abundantly applied. Feeding by means of esophageal tubes cannot be recommended, for the passage of such a tube is of as much discomfort to the patient as the act of swallowing.

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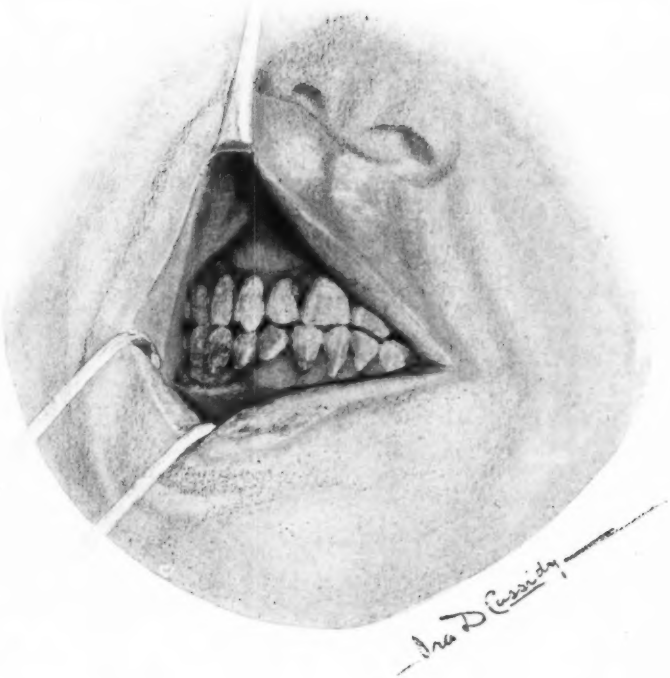

FIG. 1.—Early stage of Tuberculosis of the Gums.

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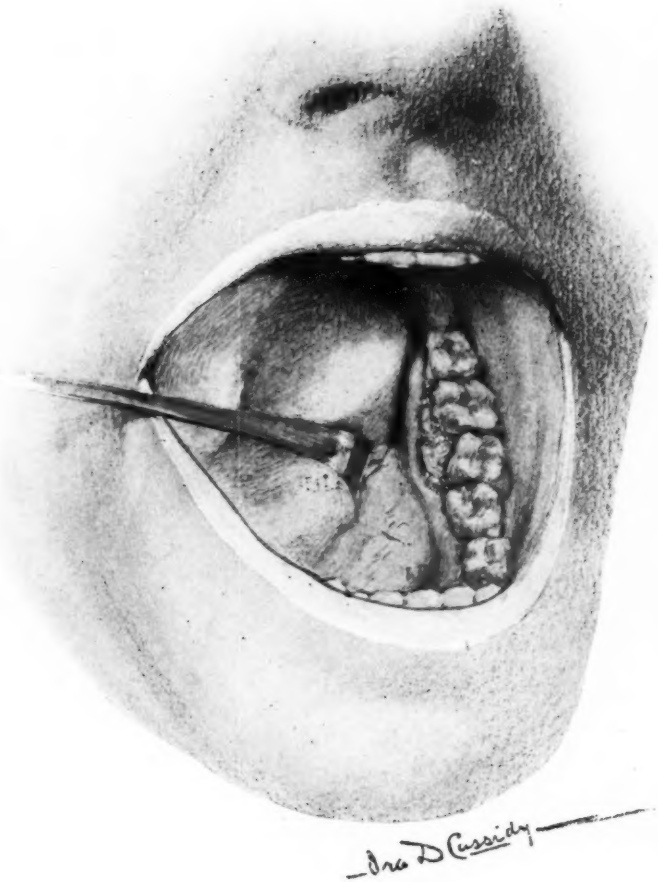

FIG. 2.—Moderately advanced Tuberculosis of the Gums.

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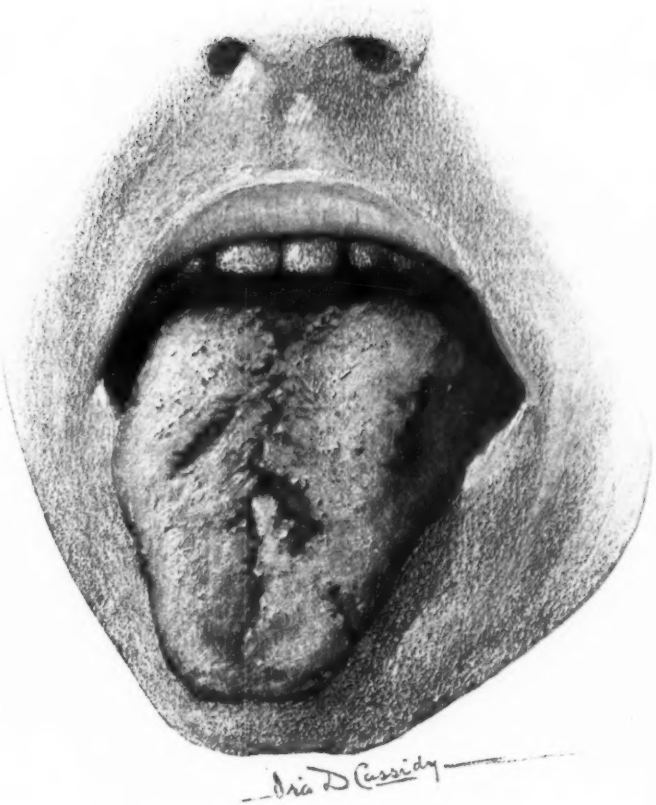

FIG. 3. Tuberculosis of the Tongue Fissured appearance, result of healing process.

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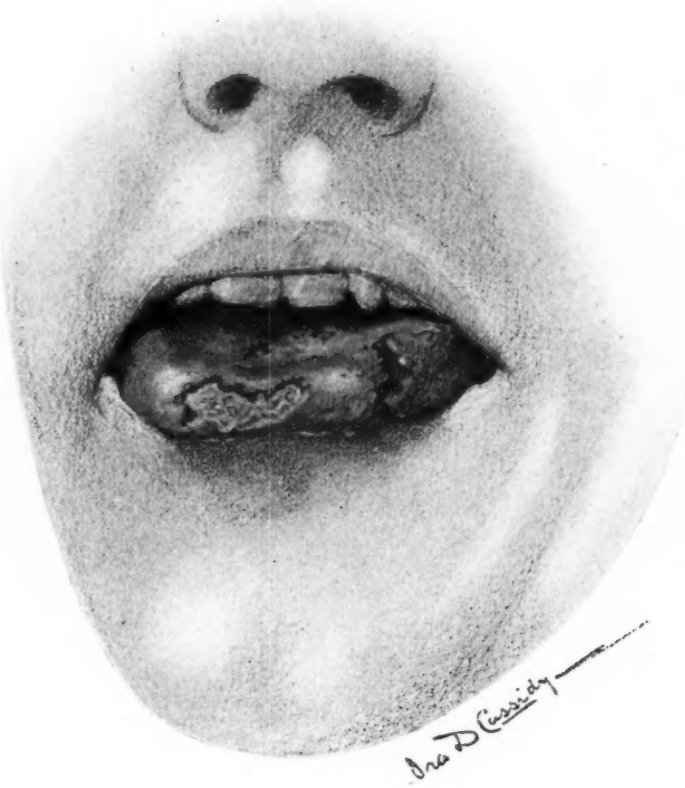

FIG. 4.—Active Tubercular Ulcer of the tip of the tongue.

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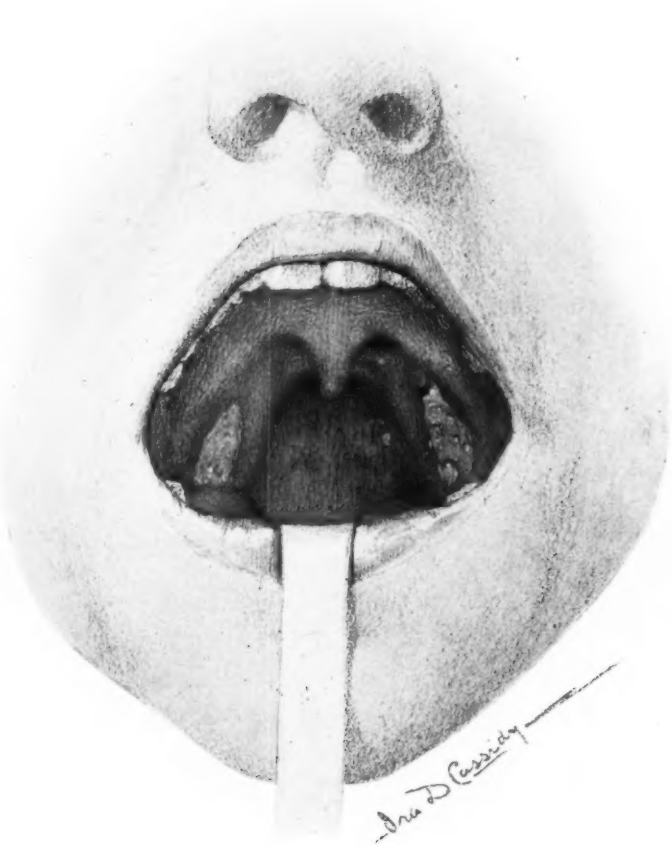

FIG. 5.—Early stage of Tuberculosis of the Tonsils.

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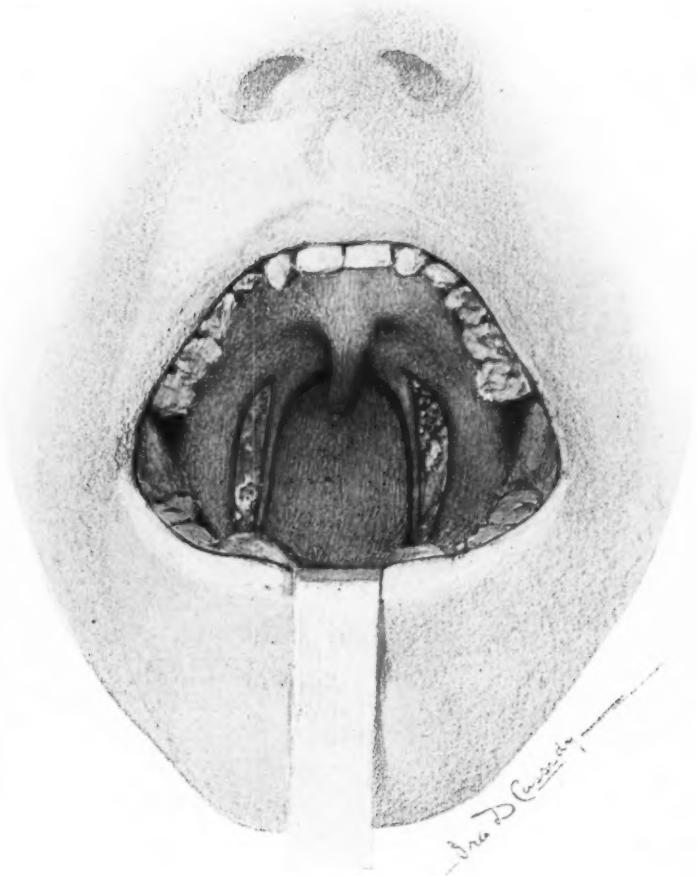

FIG. 6.—Demonstrating difference between typical Tuberculosis (left tonsil) and nontubercular exudate (right tonsil.)

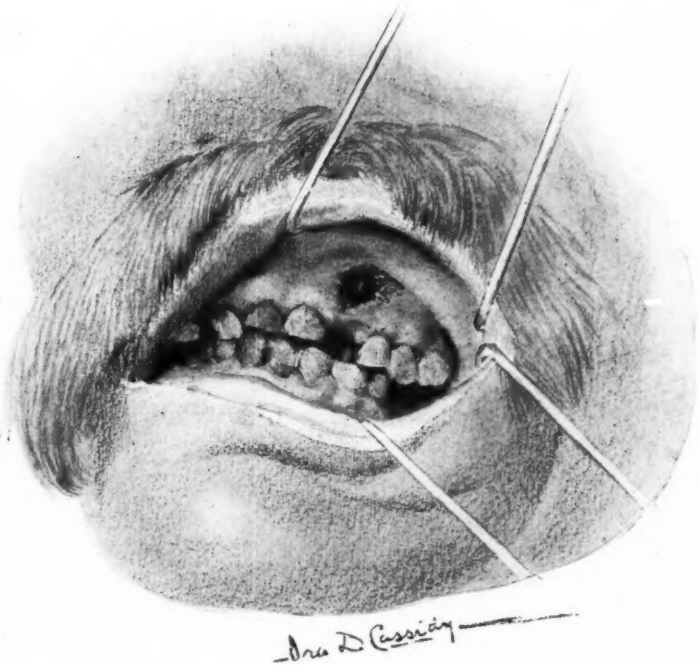

FIG. 7.—Deep Tubercular Ulceration of Gum and Alveolar process, advanced stage.

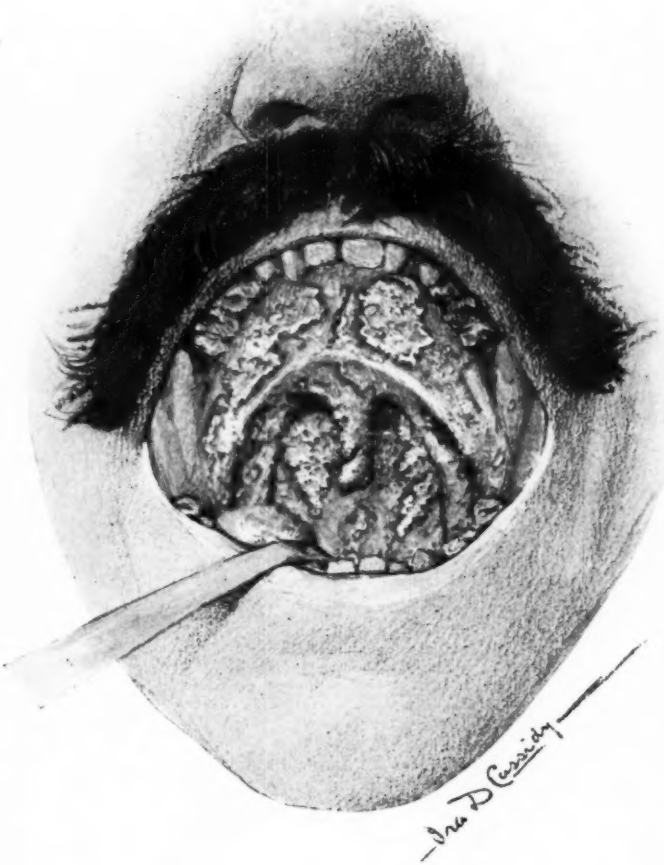

FIG. 8.—Extensive and advanced Tubercular Ulceration of the soft palate.

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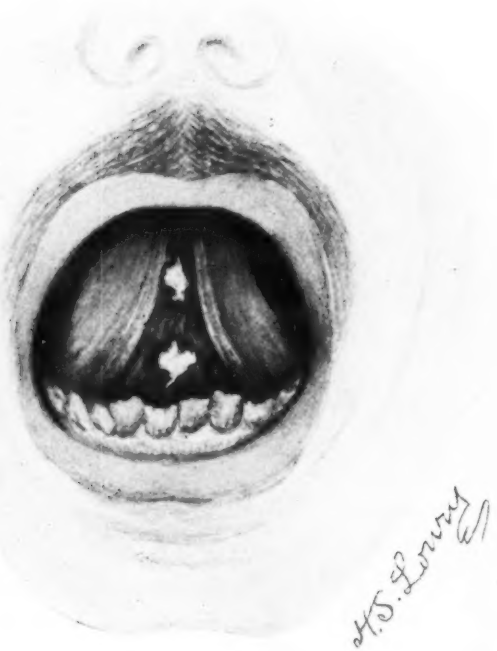

FIG. 9.—Non-tubercular Ulcerations of Fraenum in Tuberculous patient.

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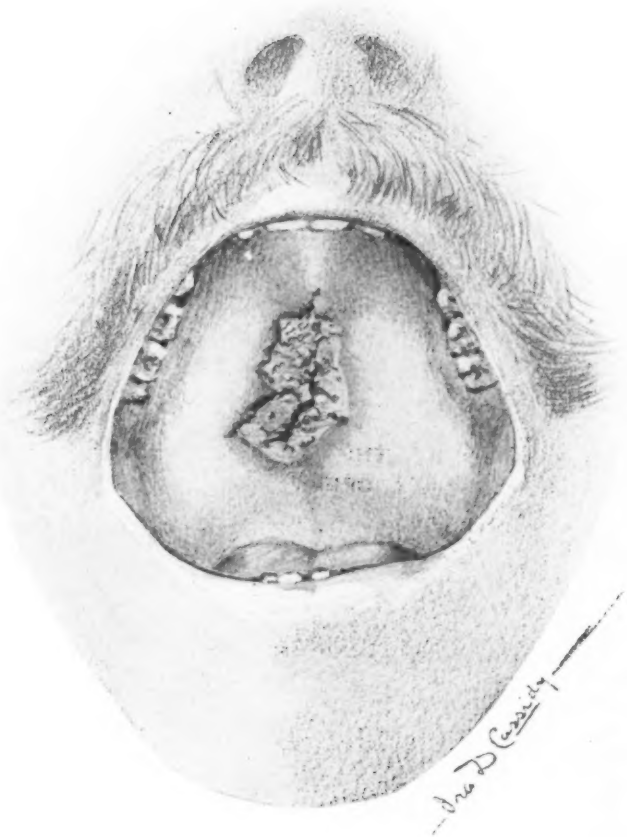

FIG. 10.—Typical Tubercular Ulceration of the hard palate.

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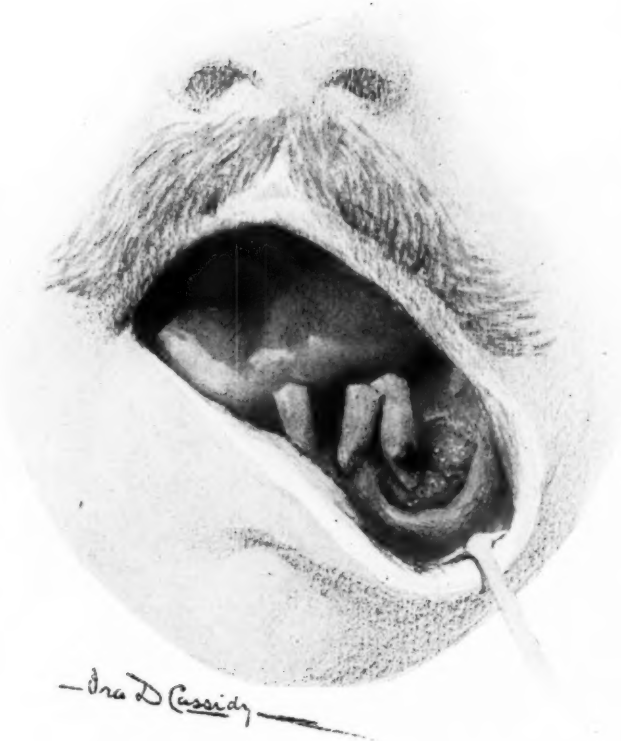

FIG. 11.—Advanced Tuberculosis of the gums.

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XLIX.

NEW METHODS OF EXAMINATION OF THE SEMICIRCULAR CANALS AND THEIR PRACTICAL SIGNIFICANCE.

By Dr. Robert Barany,

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This article treats of the practical value which the investigations in regard to the nature of nystagmus have for the otologist, the ophthalmologist and the neurologist. The subject is a hard one to understand, partly because of the difficulty of comprehending the physiologic and pathologic functions of the vestibular apparatus, and partly because this subject has not yet been treated in the textbooks devoted to otology, ophthalmology and neurology.

First, some anatomic data. The vestibular apparatus consists of three semicircular canals with their ampullae, the utriculus and the sacculus.

Nothing definite has yet been ascertained concerning the physiologic functions or the symptoms produced in diseases of the utriculus and the sacculus. The semicircular canals have long been considered the organs for the maintenance of the equilibrium of the body, but nothing positively definite has yet been ascertained to substantiate this theory. All statements in regard to this are based on hypothesis only.

The statements now to be made are facts based on observations made upon hundreds of healthy and diseased persons.

If a person sitting with head upright, on a revolving chair, is turned to the right, there will be noticed a horizontal nystagmus to the right. As soon as the turning is stopped there will be noticed a horizontal rhythmical nystagmus to the left side. This nystagmus is produced by irritation of the horizontal semicircular canals.

The experiment which directly proves this relation between irritation of the horizontal canal and horizontal nystagmus was performed by Professor Ewald in 1892 in Strassburg. He

dissected out the right bony horizontal canal of the pigeon. In the anterior part of the canal he made a little hole and over this fixed a capillary glass tube, within which was a very small glass rod. To the glass tube he attached a rubber tube with bulb. When he compressed the bulb the small glass rod compressed the fluid in the membranous horizontal canal, producing a certain movement of the endolymph. At the same moment there appeared a horizontal nystagmus in the eyes of the pigeon. (An analogous experiment can be made in men in cases where there is a fistula in the bony wall of the labyrinth.) By compression and rarefaction of the air in the external meatus, nystagmus can be produced. By the aid of this procedure, much assistance can be obtained in cases of suspected fistula of the labyrinth.

The rotation of the body on its vertical axis produces movements of the endolymph in the membranous semicircular canals.

The form and direction of the nystagmus produced by rotation depends upon the position of the head during rotation. The distant laws which govern this are more fully described in my monograph on nystagmus.*

If the semicircular canals on both sides are destroyed by disease of the labyrinth, rotation produces no nystagmus. If only one labyrinth is destroyed, nystagmus will be produced by rotation but directed more strongly to the sound than to the diseased side.

In order to make a positive diagnosis of unilateral destruction of the semicircular canals, another method is required, and this I have found. This is known as the method of diagnosis by syringing with hot and cold water. It has long been known that syringing the ear with cold water produces vertigo and nystagmus.

I have made examination of several hundred people and found that in every man with a normal vestibular apparatus, syringing the ear with cold water produces a distinct form of nystagmus, which lasts from half a minute to three minutes. If the right ear, the head being in the upright position, is syringed with cold water, there is produced a rotary and horizontal nystagmus directed toward the left side and is best

*An English translation of the new edition of Dr. Barany's monograph "Untersuchungen über den vom Vestibularapparat des Ohres reflektorish ausgelösten rhythmischen Nystagmus und seine Begleiterscheinungen" is in preparation and will be published early in 1908. The reader interested in the subject will find the complete monograph well worth study.

observed when the patient looks to the left side. If the water is of the temperature of the body, no nystagmus is produced. This reaction depends entirely on the relation between the temperature of the water and the temperature of the body. The pressure used in syringing has no influence in this connection. If the drum is intact the water must be colder than if there is a perforation, in which latter case the water comes into direct contact with the labyrinth wall, as in middle ear suppuration with large perforation or nearly total absence of the drum. In such cases you often get reaction with water of 35° C. If water of a temperature higher than that of the body is used in syringing, a nystagmus is produced which is directed toward the syringed ear, i. e., just the reverse of that produced by cold water. This reversal of the nystagmus is explained as follows: Imagine a closed cup filled with water of 37° C. to represent the labyrinth. Syringe one side of this cup with cold water. By this means the water which is near to this wall will be cooled and thereby get a greater specific weight than the other and tends to move in direction toward the bottom of the cup, while on the opposite side the warm water rises. In the same way and for the same reason there is produced a movement of the endolymph in the semicircular canal, and this is the cause of the nystagmus. It is self-evident that syringing with hot water will produce exactly the opposite effect and hence will reverse the nystagmus.

It is also self-evident that if the cup be turned 180° the movement of the endolymph is reversed. For the same reason the nystagmus is reversed when the head is directed to the floor.

If the semicircular canals are destroyed, syringing with cold water has no effect. In every middle ear suppuration, it is of great importance to know whether the labyrinth is involved in the suppurative process or not. This syringing with cold or hot water gives a precise answer in many cases where before the diagnosis would have been impossible.

The diseases of the semicircular canals can be divided into three groups.

I. Circumscribed labyrinthitis. Here the canals are not destroyed but their nerve endings are irritated by hyperemia or toxic edema. This irritation causes spontaneous nystagmus. Syringed with cold water these cases all show a typical reaction. Many diseases produce this form of nystagmus. The most important are circumscribed labyrinth suppuration, labyrinth fistula.

II. To the second group belong all the cases where the labyrinth is suddenly paralyzed. This is especially the case in acute diffuse labyrinth suppuration. In this second group syringing the diseased ear produces no nystagmus, but the sudden paralysis of one labyrinth acts in the same way as an irritation of the sound labyrinth and in such cases strong spontaneous nystagmus is manifest (directed to the sound side.)

III. The third group consists of those cases in which the destruction of the inner ear has been produced very slowly and in such a way that neither vertigo nor nystagmus has occurred, and also of cases where the destruction of the labyrinth has been of long duration. In these cases there is no spontaneous nystagmus. Syringing with cold water also gives no reaction.

In the first group spontaneous nystagmus is caused by irritation of the nerve endings of the diseased labyrinth. In these cases the nystagmus is directed to the diseased side.

In the second group, as the sudden paralysis of the diseased labyrinth acts like an irritation of the sound labyrinth the nystagmus is directed to the sound side.

Summing up in tabular form:

GROUP I.

Spontaneous nystagmus to diseased side. Fistula symptoms by compression and rarefaction of air in the external meatus.	**Reaction with cold water positive.**	**Circumscribed labyrinth suppuration; labyrinth fistula.**

GROUP II.

Spontaneous nystagmus to the sound side.	**Reaction with cold water negative.**	**Diffuse acute labyrinth suppuration.**

GROUP III.

No spontaneous nystagmus.	**Reaction with cold water negative.**	**Old diffuse labyrinth suppuration.**

In the first group the nystagmus is not continuous but occurs only from time to time and is specially produced by distinct movements of the head. Bending the head to the diseased side, backward or downward or rotating the head rapidly in the direction of the diseased side produces nystagmus and vertigo. In the interval there is little or no nystagmus.

In the second group the nystagmus directed to the sound side is continuous for two days. It is also present in the horizontal position and becomes stronger on movement. It diminishes spontaneously day by day, becomes intermittent, and after a week or fortnight almost or quite disappears. It then belongs to the third group.

Examination of the older annual reports of any ear clinic will show that a number of patients died of meningitis after the simple radical mastoid operation, patients who before the operation were strong and healthy except perhaps for occasional attacks of vertigo. The post-mortem of such patients always showed the presence of a previously undiagnosed labyrinth suppuration, which, after the radical operation, produced the fatal meningitis.

Labryinth suppuration is surely the most frequent cause of post-operative meningitis. Deafness alone is not sufficient ground for the diagnosis of labyrinth suppuration. In many cases of deafness the semicircular canals react normally to cold water and neither vertigo nor spontaneous nystagmus has occurred. The diagnosis of labyrinth suppuration is in many cases, especially in the third group, when no spontaneous nystagmus is present, impossible without investigating the reaction with cold water.

The diagnosis of labyrinth suppuration being made, every case of such suppuration should be operated on radically and the labyrinth itself opened at the same time.

Since we have been opening the labyrinth in every case of labyrinth suppuration we have not had any post-operative meningitis. The operation is indeed difficult but we have lost no patients as a result of the operation, although during this year we have operated on more than thirty patients. All the patients have been operated upon after the method of Neuman.

Not only for the diagnosis and treatment of labyrinth suppuration is the consideration of nystagmus very important but

also for the diagnosis of cerebellar abscess and cerebellar tumor, or tumor of the nervus acusticus.

The nystagmus in these cases is produced by irritation or paralysis of the nervus vestibularis or of the nucleus in the medulla oblongata or of Deiters' nucleus.

The diagnosis of cerebellar abscess in middle ear suppuration is very difficult, and I have seen some cases where the nystagmus alone has helped us to the diagnosis with resulting operation and cure of the patient. Most cerebellar abscesses are caused by an old labyrinth suppuration, and in these cases the diagnosis is relatively easy. If one has to deal with a chronic middle ear suppuration without fever, with deafness, and no reaction to cold water, and this patient has strong spontaneous rotatory nystagmus to the diseased side, the diagnosis of cerebellar abscess can be made at once and for the following reasons: If the patient does not react to the syringing with cold water, the labyrinth must be destroyed and he can have spontaneous nystagmus to the sound side or no nystagmus. Spontaneous rotatory nystagmus to the diseased side proves that this nystagmus is produced by an intracranial complication in the posterior fossa of the skull, and as there is no fever and no meningitis there must be a cerebellar abscess. Tumors of the cerebellum or of the acoustic nerve produce analogous symptoms.

In many cases of deafness, normal drum, no reaction to cold water, strong rotatory nystagmus to the diseased side, the diagnosis of acoustic tumor can be made even before the patient gets a paralysis of the facial nerve, of the trigeminus, papillitis optica, etc.

There is another class of patients in which the determination of the nystagmus will be of value. These are cases of trauma of the head, with lesion in the inner ear. Nearly all the cases with cerebral concussion complain of vertigo and disturbances of equilibrium. As a result of the investigation one always finds an organic defect with a traumatic neurosis. The knowledge of nystagmus will help one to distinguish between the two. As regards the nystagmus these cases belong to the second group, have fits of vertigo occasioned by irritation of the nerve endings in the ampullae. These attacks occur without any apparent cause or in connection with the distinct movement of the head previously mentioned, viz.: bending or turning the head to the side, bending the head down-

ward or backward, sudden rising from the horizontal to the vertical position, and quick walking. It is very important to know that these movements do not always produce vertigo or nystagmus; if the patient has had vertigo just before there will be a long pause before vertigo or nystagmus will again be produced. If strong nystagmus is observed there is organic lesion. Many of these patients not only have vertigo but also nausea and vomiting, and it may be, partial loss of consciousness. These concomitant symptoms are of neurotic nature. Also, the disturbances of equilibrium are mostly neurotic. Disturbances of equilibrium in connection with nystagmus must have a distinct character; they are determined by the form and direction of the nystagmus and the position of the head. If there is rotatory nystagmus to the right side, the patient will fall to the left side if the head is upright. If the head is turned to the left he will fall forward. If the head be turned to the right he will fall backward. In cases of traumatic neurosis, disturbances of equilibrium do not follow these rules or are exaggerated. The falling is in no definite direction, to the right, the left or backward like a stick, and turning the head has no influence on the direction in which he falls. The knowledge of the phenomena of nystagmus incident to these cases will enable one to determine how much of the phenomena of which the patient complains is of organic or neurotic origin.

L.

CIRCUMSCRIBED PURULENT INFLAMMATION OF THE LABYRINTH.*

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Translated With the Author's Permission.
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Previous discussions regarding purulent inflammation of the labyrinth have had to do principally with the diffuse inflammations, while the circumscribed labyrinthine inflammations have been but briefly touched upon. According to our experience based upon a large amount of clinical material, it is the circumscribed inflammations of the labyrinth which should be considered of the first importance. My statistics to which I shall later refer in detail assuredly prove that circumscribed inflammations of the labyrinth are not only of the greatest prognostic significance but also of the greatest weight as a point of indication for the operative opening of the labyrinth.

From the clinical standpoint, we divide the purulent inflammations of the labyrinth arising from acute and chronic purulent middle ear diseases into the following classes:

1. Diffuse and circumscribed.
2. Manifest and latent.

An exact test of the condition of the labyrinth is of fundamental importance before doing any middle ear operation. The hearing tests are made with the tuning fork, speaking tube, the tests of Lucae-Dennert and Stenger, and the continuous tone series of Bezold, while the excitability and condition of the vestibular apparatus are tested with hot and cold syringing, rotation of the body and compression and expansion of the air in the external canal, etc. The condition

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of the labyrinth thus revealed gives, as a rule, a true and reliable picture of the pathologic conditions in the interior of the labyrinth and renders possible an exact localization of the same in its individual portions.

Especially is the fundamental testing of the vestibular apparatus of importance for further action and for the place of operation upon the labyrinth.

Manifest, circumscribed disease of the vestibular apparatus shows itself by sharply marked clinical symptoms (vertigo, disturbance in maintenance of body equilibrium, nystagmus and the like), while the latent purulent inflammations in the region of the vestibular apparatus run their course without symptoms and can be diagnosed only with the aid of an exact testing of the labyrinthine functions.

It is therefore clear that following the complete radical mastoid operation, such a circumscribed latent purulent inflammation may become manifest and produce a fatal postoperative meningitis.

If, however, the functions of the labyrinth are carefully tested before the radical operation and the possibility of circumscribed purulent inflammation considered in our calculations, we can with certainty prevent such fatal accidents. As a matter of fact, in our clinic, in all those cases where the above mentioned points were sufficiently determinative we have had no deaths to deplore.

It is to be stated emphatically that for the recognition of circumscribed purulent areas, it is the test of the function of the vestibular apparatus to which we must specially direct our attention.

For the most part the hearing test leaves us in the lurch. In by far the greater number of cases, we are unable to say whether the difficult hearing or absolute deafness which we discover is the result of purulent inflammation in the labyrinth or in the middle ear. Only in those cases where a high degree of deafness or absolute deafness appears suddenly can we with certainty locate it in the labyrinth.

Under the term manifest diffuse purulent inflammation of the labyrinth, whether occurring in course of acute or chronic middle ear suppuration, I include all those cases where besides the sudden appearance of a high degree of deafness or absolute deafness, clearly defined vestibular symptoms are present. These symptoms manifest themselves in spontaneous nystagmus toward the sound side (through a failure of function of the

vestibular apparatus of the diseased side or through an overbalancing of the sound side), and in very much lessened sensibility, even to absolute loss, of the vestibular apparatus of the diseased side, together with dizziness, vomiting and disturbance of equilibrium.

In diffuse latent purulent inflammation of the labyrinth, there is neither disturbance of equilibrium, vertigo nor nystagmus, and the presence of such an inflammation can be supposed only when, besides the sudden appearance of absolute deafness or a high degree of loss of hearing stated by a patient, the testing of the vestibular apparatus shows complete loss of its function or a high degree of diminution of its irritability.

The circumscribed purulent inflammations of the labyrinth in the same way can be divided into manifest and latent. They affect either the cochlea or the vestibule. Occasionally they are limited to a circumscribed portion of the cochlea or the vestibule.

Before proceeding to consider these last, I will briefly describe those transitory labyrinth symptoms that not infrequently follow middle ear operations. They are caused by increase in the labyrinthine pressure, either by retention of pus in the middle ear, or too tight pressure from gauze wicks and the like, or by ostitis of the pyramid without disease of the labyrinthine contents. They disappear rapidly and may maintain their physiologic reactions.

The circumscribed purulent inflammations of the labyrinth may be localized either in the cochlea or the vestibular apparatus; occasionally they affect only a narrow, sharply defined area. Of my 52 cases of purulent labyrinthitis, seventeen (32.7 per cent) were circumscribed. The circumscribed forms can be divided into manifest and latent. For the determination of the very necessary information, whether in the individual case we have to do with the manifest or latent circumscribed purulent labyrinthitis, we can depend only upon the result of the test of the functions of the vestibular apparatus. For example, if there is present hearing and spontaneous nystagmus to the diseased side, while the examination of the irritability of the corresponding vestibular apparatus shows it still present, then we have to deal with the manifest limited purulent inflammation of the vestibular apparatus, in case the fistula symptom is present or the radical operation dis-

closes the presence of a fistula. If, on the other hand, fistula and fistula symptoms are absent, there remains merely disease of the labyrinth capsule. At the same time there are present the known symptoms of vestibular disease, such as dizziness, disturbances of equilibrium, ataxia and the like. If, on the other hand, these symptoms completely fail, and the hearing function is maintained, while the functional tests of the affected vestibular apparatus shows a great diminution or absolute failure of excitability, then we have to deal with the circumscribed latent purulent inflammation of the vestibular apparatus.

Much more difficult are the conditions when we have to deal with a circumscribed purulent inflammation of the cochlea. If we meet with total labyrinthine deafness in the presence of maintained vestibular function, the next thing which must be searched for is a basic point which can be used in disturbance of the hearing due to purulent inflammation of the cochlea. It is necessary in this connection to know the clinical history. If the patient relates that the complete deafness appeared suddenly and was accompanied with vestibular symptoms, as vertigo, disturbances of equilibrium, nausea, vomiting and the like, then (in so far as the functional tests show the vestibular apparatus to be in normal condition) we can assume a circumscribed purulent disease of the cochlea to be present. This supposition acquires greater probability in cases where the laying bare of the middle ear cavity reveals the presence of the labyrinth fistula.

As regards the term labyrinth fistula, I would state that in common with Hinsberg, Wittmaack and others, I use the word fistula only when a purulent canal leads from the interior of the labyrinth outwards or conversely from the middle ear to the labyrinth. Superficial erosion from pressure limited to the bony labyrinthine capsule or purulent softening is in the anatomic sense no fistula. It is on the other hand to be called a defect in the labyrinthine wall or a defect in the semicircular canals. As a rule, such defects in the bony wall act as a point of lessened resistance for the membranous labyrinth, because through the breaking down of its bony protecting wall, traumatic and inflammatory injuries are easily received. In the presence of inflammatory processes within the middle ear, such a labyrinth is in a condition of increased excitability, so that even a slight irritation is capable of setting

up serious vestibular symptoms. Sometimes a slight mechanical irritation, as too tight packing of cotton, increased or diminished air pressure, produces sufficient irritation to bring about severe symptoms in the vestibular apparatus.

Although, as appears from the foregoing, there is a good deal of difficulty in the making of the diagnosis of a defect in the labyrinth capsule, it is nevertheless a valuable supporting fact in the interpretation of the clinical appearances. If for example, we have the history of a suddenly appearing absolute deafness and the radical operation shows a defect in the labyrinth wall, the pathologico-anatomic substratum of which cannot be determined on the living with certainty, nevertheless this defect can with the greatest probability be considered as a fistula and the clinically produced absolute deafness as due to a purulent inflammation of the cochlea. The same applies as a matter of course to defects or fistulae of the semicircular canals.

The points of view just developed were determinative for me as furnishing the indications for opening the labyrinth, although we must emphasize the fact that precise indications can be established only from the relationship between the clinical symptoms taken in connection with the findings at operations.

Tabulated indications as given by me include only the main types of purulent disease of the labyrinth as, on account of the many variations in the clinical picture, a complete classification of all the appearances is hardly possible. I can, however, express the conviction, based on a large clinical experience, that this table is of value in every case. I will now briefly consider the individual indications.

1. The hearing and vestibular functions are intact and the total radical operation shows the presence of a fistula. As regards the intactness of the labyrinth function, it must be taken into consideration that in this case there is not a real fistula but a defect in the labyrinthine wall. If spontaneous nystagmus appears, it is due to an irritation of the vestibular apparatus from hyperemia, increased pressure and the like. Since it is a case of a complicated middle ear process, the opening of the labyrinth without regard to the presence of spontaneous nystagmus is contraindicated. I recall eight such cases, in two of which, it is true, the labyrinth operation was done. In one case there appeared severe meningeal

symptoms; in the second a deep extradural abscess gave the indication for the opening of the labyrinth—both cases recovered.

2. Hearing gone, vestibular apparatus functionating (irritable) and the radical operation shows a fistula. If in this case there is any spontaneous nystagmus, we have to deal with a circumscribed purulent disease of the vestibule. If at the same time there is present increasing temperature with meningeal symptoms—the opening of the labyrinth is indicated. On the other hand, this is not to be done if the temperature is normal. The labyrinth operation is contraindicated if spontaneous nystagmus is absent. My statistics cover ten such cases, of which eight had spontaneous nystagmus. The labyrinth operation was done six times with good results. One patient died of tubercular meningitis, the other of acute purulent leptomeningitis. In the last case, for other reasons, only a simple opening of the mastoid was done. The remaining two cases without spontaneous nystagmus healed completely after the radical mastoid without delay.

3. Hearing present, vestibular apparatus does not react, and the radical operation shows a fistula. In such cases there is no doubt that we have to deal with a circumscribed purulent disease of the semicircular canals and the labyrinth operation is indicated in the presence of the other accompanying symptoms, especially increasing fever. As regards the indication for operation, the presence or absence of spontaneous nystagmus does not come into consideration. My statistics cover two such cases, which had spontaneous nystagmus. In one case the labyrinth operation was undertaken. The first case was a circumscribed purulent inflammation of the semicircular canal for which a radical operation was done. In the second case, the increase of temperature gave the indication for the opening of the labyrinth.

4. This group includes those cases where the hearing function is present, the vestibular apparatus does not react, but no labyrinthine fistula is found at the radical operation. If we consider that, aside from this, the symptoms indicate purulent disease of the labyrinth and especially if there is increasing temperature, then we perform the labyrinth operation immediately after the radical operation. This applies also to those cases where the laying bare of the middle ear area uncovers other complications, such as deep seated extradural

abscess, cerebellar abscess and the like. I have observations concerning two cases belonging to this class. In the one case there was a deep extradural abscess whose point of origin, based on previous testing of the labyrinth, must have been in the vestibular apparatus. I accordingly did the radical operation and the labyrinth operation at the same time, and the further history of the cases showed the correctness of the method of procedure. Several days after the opening of the labyrinth, there appeared the symptoms of a cerebellar abscess which was evacuated and the patient recovered. Without the previous operation on the labyrinth the diagnosis of cerebellar abscess would have been impossible, since it was based entirely on the sudden reversal of the nystagmus. Before the labyrinth operation the nystagmus was toward the sound side. In the second case, in which neither increased temperature nor other special labyrinth symptoms, with the exception of spontaneous nystagmus, were present, I refrained from opening the labyrinth.

5. Hearing power and vestibular function both destroyed; fistula in the labyrinth capsule. While this group, strictly speaking, must be classed among the diffuse purulent inflammations of the labyrinth, I will discuss it here briefly as it includes the cases of greatest practical importance. Without regard to the presence or absence of spontaneous nystagmus, the radical labyrinth operation on one side is absolutely indicated.

My own cases in this group are ten in number, in six of which spontaneous nystagmus was present. Of these six, five recovered, one died. As this last case is of especial interest and is suitable for showing most clearly the point of election for the operation, I will briefly describe it. The patient was twenty years old; there was entire loss of hearing and vestibular function, with spontaneous nystagmus towards the sound side. She showed the typical picture of manifest diffuse labyrinthitic suppuration. On laying bare the middle ear region, a fistula of the semicircular canal was found, so that according to my view the immediate radical labyrinth operation was indicated. For other reasons the labyrinth operation was not performed and the patient went on to purulent leptomeningitis. Had the labyrinth been opened at the time of operation, the patient's life would in all probability have been saved. Of the other four cases, which without spontaneous nystagmus were latent diffuse purulent laby-

rinthitis, one died of tubercular meningitis, one of pleuropneumonia, and two got well.

6. Hearing and vestibular function gone, no labyrinth fistula. If spontaneous nystagmus appears it is certain that we have to do with a manifest diffuse purulent labyrinthitis, where the fistula is not visible macroscopically. Here the one-sided opening of the labyrinth is absolutely indicated.

I have had four cases of this class with two deaths. In one case the operation had to be stopped during the laying bare of the middle ear cavity on account of asphyxia during the narcosis.

In the second case there was already present a diffuse purulent meningitis (thick fluid from lumbar puncture); the other two cases recovered. If spontaneous nystagmus is absent, then we have latent diffuse purulent labyrinthitis. Here the labyrinth operation is to be considered only under special circumstances, as fever, meningeal symptoms and the like. My statistics cover six such cases. In two cases there were sequestra of the pyramid with healing; twice I left the labyrinth operation out of consideration. In one case the sudden appearance of facial paralysis gave the indication for the labyrinth operation, and in one case the labyrinth operation was discontinued and the patient had post-operative meningitis.

7. Hearing function gone. Vestibular apparatus normal, no labyrinth fistula. In this class of cases we do not consider opening the labyrinth. In five cases observed by me there was spontaneous nystagmus in two, while it was absent in three. Of the latter, two recovered after the radical operation, one died of a symptomless brain abscess (temporosphenoidal). The two cases with spontaneous nystagmus recovered.

From the foregoing it is evident that where treatment followed the developed diagnostic points and the symptoms were fairly clear and well supported, a good result was brought about. The discovery of an already existent latent purulent process in the labyrinth is of primary importance. If we are able to ascertain this, and make the presence of a purulent focus probable, then there is strong probability of preventing the post-operative meningitis.

Of my 52 cases of infective labyrinth inflammation, in 27 the labyrinth operation was performed. Healing resulted in 20 cases; in seven, death resulted. Of the deaths, two took place from tubercular meningitis, one each of pleuropneumonia

and brain abscess, two of acute purulent leptomeningitis (in these the labyrinth, contrary to my indication, was not opened at the time of the radical operation but later), and in one case there was present at the time of admittance a diffuse purulent leptomeningitis. In no case was the labyrinth operation as such the cause of the fatal result. On the other hand, the study of my cases has given the significant result that as a rule the fatal result was due to the failure to perform the labyrinth operation at the right time. It is of great importance to find before doing the radical operation whether the opening of the labyrinth is to be done immediately following or not. In a number of cases it is impossible to determine beforehand whether the labyrinth should or should not be immediately opened, yet even here the anatomic appearances of the labyrinth wall of the semicircular canals, gives us valuable information for our further action.

Of the remaining twenty-five cases, where there was no positive indication for opening the labyrinth, an operative purulent lesion of the semicircular canals was twice found with resulting recovery.

Fifteen times a fistula was found in the semicircular canals, once on the tentorium. Fifteen cases were operated radically and one treated conservatively; all recovered. Of the ten cases where no fistula was found at the operation, nine were radically operated and one treated conservatively; all recovered.

It has not been my purpose to give all the observations which have served to give us light on this subject, or to construct a table which is to be considered as the final word on this subject, but to lay before you the principal diagnostic points. These are not the result of theoretic considerations, but of observation on a large amount of clinical material.

It admits of no doubt that future investigations will bring to pass important changes in our observations and in the views advanced by me. However, I think I am not mistaken in asserting that the points cited are of practical value and should be carefully weighed. Whenever I have been able to proceed according to the indications enumerated, there have been no bad results.

LI.

THE CONSTRUCTION OF THE ETHMOID LABYRINTH.

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Anatomically, as well as clinically, the ethmoid labyrinth occupies a central position in its relation to the other nasal accessory sinuses. Diseases of the frontal and maxillary sinuses are frequently combined with an involvement of the cells of the ethmoid. That this association is dependent on the close anatomic relations existing between the frontal sinus and the ethmoid labyrinth on the one hand and the maxillary sinus and the ethmoid labyrinth on the other, there can be little doubt.

The object of this paper is to call attention to certain fundamental facts in the construction of the ethmoid labyrinth, and to point out the close anatomic relations existing between the ethmoid labyrinth and the other nasal accessory sinuses.

The location of the ethmoid labyrinth is well shown in a cross section as in Fig. 1. The labyrinth lies external to the meatus nasi communis and internal to the orbit and the antrum. Above is the floor of the anterior cranial fossa and below is the meatus nasi media. The inferior and median walls of the labyrinth are marked by several conspicuous structures. These are, from below upward, the processus uncinatus, the bulla ethmoidalis, the concha media, the concha superior, and the concha suprema.

In a section made through the ethmoid the labyrinth is seen to be made up of a number of air spaces or cells, which vary in size and are separated from each other by thin bony plates. These plates are formed with very little regularity. They have, however, certain relations in common. In the first place, all

the plates end abruptly as soon as either the orbital wall or the upper wall of the labyrinth is reached (see Figs. 1, 2, 3), This is not the case, however, on the median aspect of the ethmoid labyrinth. Here the plates do not end abruptly, but project beyond the labyrinth and form prominences which extend into the nasal chamber (see Fig. 2). The prominences thus formed by the extension of the partition plates of the ethmoid labyrinth over in the nasal fossa are known respectively as the processus uncinatus, the bulla ethmoidalis, the concha media, the concha superior and the concha superma.

The openings for the several ethmoid cells into the nasal chambers are always found between these prominences formed by the extension of the partition plates, that is, between the unciform and the bulla, between the bulla and the concha media and between the concha media and the concha superior. This relation of the plates of the ethmoid labyrinth and the several openings of the ethmoid cells into the nasal chambers is well shown in Fig. 2. In this preparation an unusually simple, uncomplicated arrangement of the several ethmoid cells is found. Such preparations may be looked upon as representing the typical construction of the ethmoid labyrinth. The study of such a preparation will assist greatly to an understanding of what is sometimes referred to as the architecture of the ethmoid labyrinth. In the preparation shown in Fig. 2 the somewhat atrophic concha media has been partially detached. The concha superior is even smaller than usual, while the concha suprema is absent. The compartments or cells of the labyrinth have an extremely simple arrangement, owing to the fact that the several partition plates extend through not only to the orbital wall but to the upper wall of the labyrinth as well. In this preparation, moreover, but a single instance is noted where the several cells of the labyrinth communicate on account of imperfect separating plates. Usually a number of such defects in the separating plates exist and necessarily give to the labyrinth a much more complicated arrangement.

The lower or first plate of the ethmoid forms the processus uncinatus. This plate is always in a rudimentary state and only occasionally is it found forming the upper boundary of an ethmoid cell. When such a cell exists it forms a large ager nasi (see Fig. 4).

The second plate of the ethmoid is called the plate of the

bulla because the extension of this plate into the nasal chamber forms the bulla ethmoidalis. Between the processus uncinatus below and the bulla ethmoidalis above is found a depression or trough. The mouth of this trough is called the hiatus semilunaris, whereas the depression or trough itself is known as the infundibulum ethmoidale. Into this infundibulum there are several openings. In its posterior end is the opening for the maxillary sinus. The anterior end of the infundibulum opens usually into the frontal sinus. Besides the openings for these two accessory sinuses, one or more ethmoid cells frequently open into the infundibulum. These cells are called the infundibular ethmoid cells. Such cells are usually located in the unciform process (see Fig. 4) or they are cells formed by a closure of the anterior end of the infundibulum which dilates in the floor of the frontal sinus into an ethmoid cell, as shown in Fig. 5.

The third partition plate of the ethmoid labyrinth is the plate of the concha media (see Fig. 2). Between the second and third ethmoid plates, that is, between the plate of the bulla and the plate of the concha media, is an opening usually quite large, leading into the ethmoid labyrinth. This is the opening for the anterior, or, as they are sometimes called, the middle ethmoid cells.

The fourth partition plate is the plate of the concha superior. Between the extension of the third and fourth plates, that is, between the concha media and the concha superior, is the meatus nasi superior. Into this meatus there are usually several openings leading from the ethmoid labyrinth. These are the openings for the posterior series of ethmoid cells.

The fifth partition plate is the plate of the concha suprema, and like this structure, it is often entirely wanting. In the preparation shown in Fig. 2, the plate of the concha suprema is developed, although the extension of this plate, the concha suprema, is itself scarcely detectable. The plate in this preparation, however, is imperfectly formed, allowing free communication between the ethmoid cells on either side of it.

This arrangement of partition plates and openings for ethmoid cells represents what is understood as the typical or fundamental architecture of the ethmoid labyrinth. The great complexity in the construction of the ethmoid, which has given rise to the term labyrinth, is the result in the first place of

extreme irregularity in the formation of the partition plates, and in the second place of the occurrence of defects in the several partition plates, allowing of communication between cells belonging to the different groups, that is, between cells of the anterior and posterior ethmoid series. This irregularity in the formation of the partition plates results at times in producing a large number of small cells, and at other times in producing extremely large ethmoid cells, so that occasionally the whole ethmoid labyrinth consists of but a couple of very large ethmoid cells. This same irregularity in the formation of the plates of the ethmoid results sometimes in carrying an ethmoid cell belonging to the anterior series well back to the posterior end of the labyrinth. In this case the opening being found between the bulla and the concha media in the meatus nasi media, shows clearly that the cell must be classed as an anterior or middle ethmoid cell. On the other hand, a posterior ethmoid cell with its opening in the superior meatus, that is, between the concha media and the concha superior, is sometimes pushed so far forward as to produce a prominence in the floor of the frontal sinus.

Still another result of the irregular formation of the ethmoid labyrinth is the tendency for ethmoid cells to develop outside the usual confines of the labyrinth. Such anatomic variations are by no means uncommon, and when met with clinically or post-mortem their true nature has often been misunderstood. For example, an ethmoid cell is sometimes pushed back into the sphenoid sinus where it occupies the upper part of this cavity. This has sometimes been wrongly interpreted as representing a double sphenoid sinus. More frequent is the invasion of the frontal sinus by an ethmoid cell. Here, too, the wrong interpretation has often been given that a double frontal sinus exists. This is especially likely to be the interpretation when the ethmoid cell which has pushed into the frontal sinus is a large one.

An ethmoid cell which has developed at the expense of the frontal sinus is known as a bulla frontalis. It may have its origin in one of three ways: First, by an anomaly in the plate of the unciform process, by which an ethmoid cell, developing in front of the process, pushes this plate up into the frontal sinus, as shown in Fig. 6. Second, by the development of an ethmoid cell in the closed anterior end of the infundibulum, as

shown in Fig. 5. This cell may expand in the floor of the frontal sinus and produce a bulla frontalis. The third way in which the bulla frontalis is known to form is when the plate of the bulla ethmoidalis is pushed forward into the frontal sinus. Examples of ethmoid cells which have invaded the frontal sinus are shown in Figs. 4, 5, 6, 7. These cells are found at times almost completely filling the frontal sinus.

The occurrence of an ethmoid cell in the anterior end of the concha media is quite common. Occasionally such a cell becomes very large, producing a cystic enlargement of the anterior end of the turbinated body (see Fig. 7). This condition is known as a concha bullosa. That these cystic enlargements of the concha media represent but an enlarged ethmoid cell which has expanded in the turbinated body, has often been overlooked, and some rather interesting hypotheses have been advanced to explain their origin. Some have considered this condition to be the result of a "rarefying ostitis." By others these cysts are supposed to be caused by a curling under of the free edge of concha media which in turn becomes attached along the base of the turbinated body in the middle meatus. The occasional large size of the cyst is then explained as the result of a dilatation of this cavity from its filling with mucus. Others have looked upon these cysts as ethmoid cells that have expanded and been pushed out into the turbinated body on account of the existence of a closed empyema of the ethmoid labyrinth.

The real nature of these cysts of the concha media is undoubtedly the same as of the ethmoid cells which invade the frontal or the sphenoid sinuses. They represent an anatomic variation in the form of a large ethmoid cell growing out into the concha media. This cell, like any of the ethmoid cells, may occasionally become the seat of a mucocele or an empyema. The existence of the concha bullosa is not in any way the result of the mucocele or the empyema.

The cells of the ethmoid labyrinth are in close relation to the orbit. In the first place, many of these cells are separated from the orbit merely by the thin orbital or paper plate of the ethmoid. This relation is shown in Fig. 3. Again it is not uncommon to find ethmoid cells which extend out along the upper wall of the orbit. These supraorbital cells of the ethmoid come necessarily into close relation with the orbital extension of the frontal sinus, the two being separated by a

thin partition of bone. If a sagittal section be made in such a case, through the frontal sinus and the orbit, this plate separating the orbital ethmoid cells from the orbital extension of the frontal sinus will appear in cross section (see Fig. 8). Sections showing this condition have frequently been described as illustrating the existence of compartments in the orbital extension of the frontal sinus, whereas such compartments usually represent supraorbital ethmoid cells.

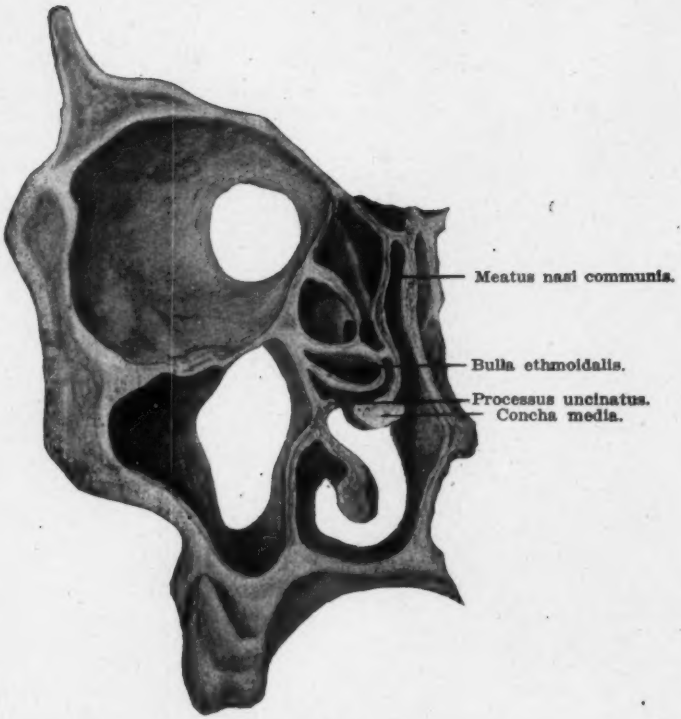

Fig. I.

Frontal section showing right side of nose. Cross section through the ethmoid labyrinth.

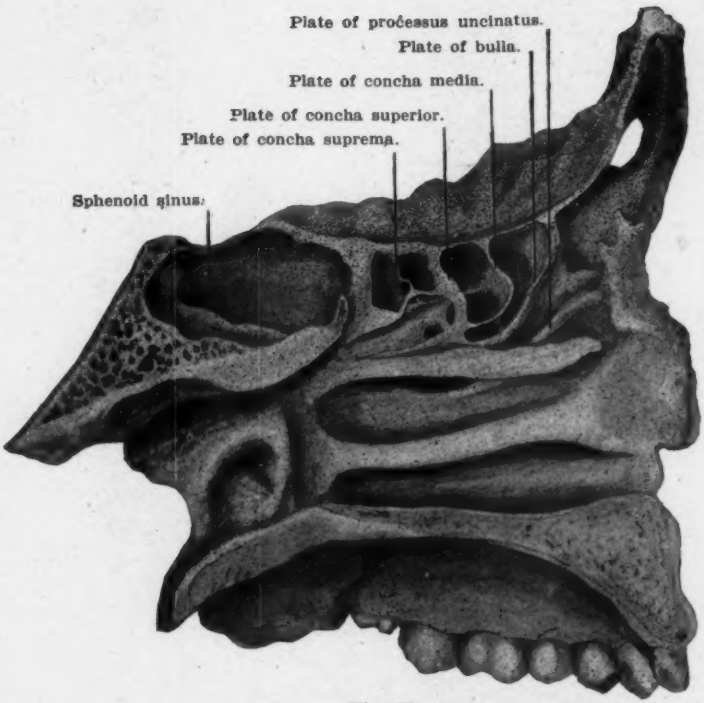

Fig. II.

Sagittal section showing left side. Preparation shows typical construction of ethmoid labyrinth; the several partition plates presenting an unusually simple form.

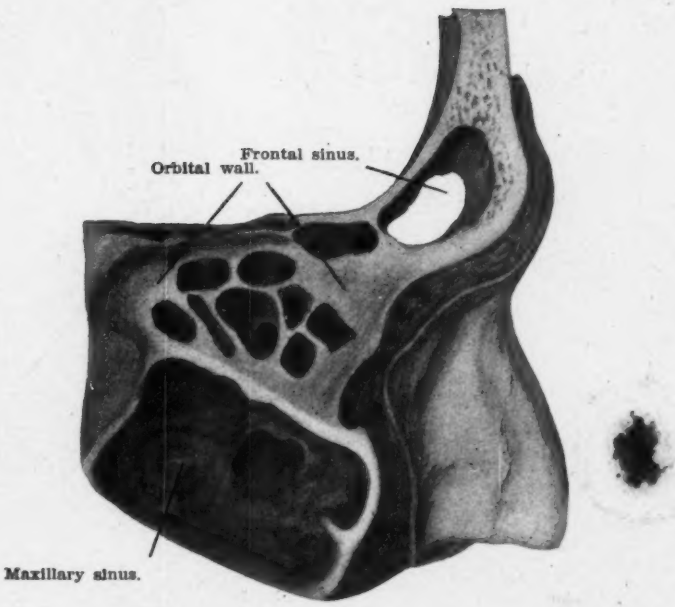

Fig. III.

Sagittal section through the orbit, section opening into the frontal sinus and maxillary sinus. The several ethmoid cells which impinge upon the paper plate of the ethmoid have been opened so as to show the relation between the cells and the orbit.

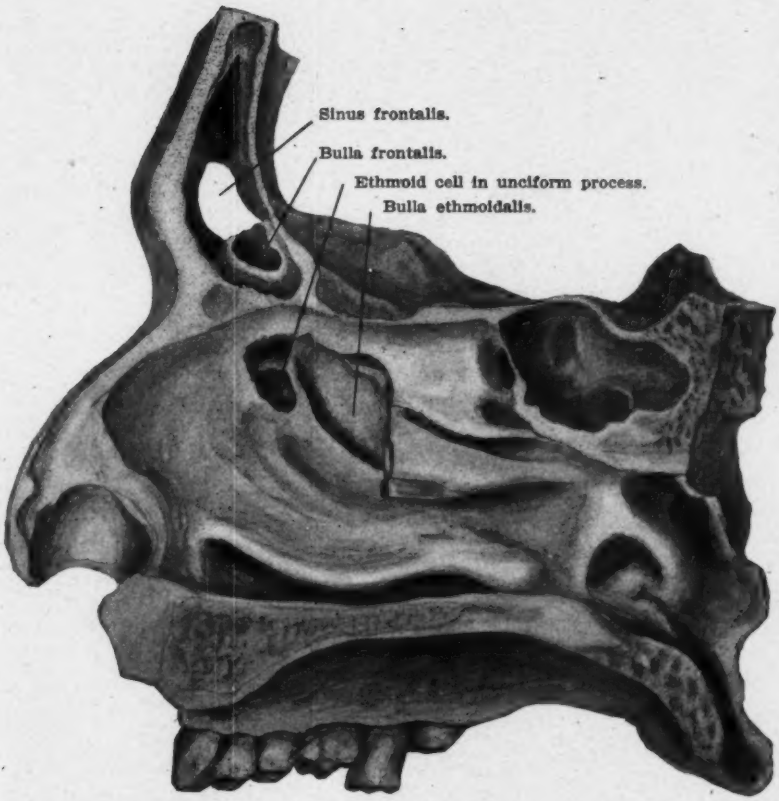

Fig. IV.

Sagittal section showing right side. The anterior half of middle turbinated body has been removed. The preparation shows a large ethmoid cell, which has been developed in the floor of the frontal sinus; also ethmoid cell in the unciform process.

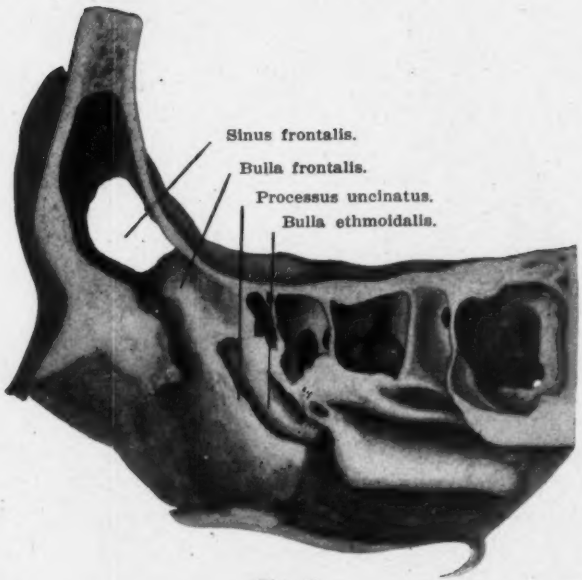

Fig. V.

Sagittal section showing right side; the anterior half of the middle turbinated body has been removed; the infundibulum, instead of opening into the frontal sinus, opens into an ethmoid cell which has developed in the floor of the frontal sinus; the opening into the frontal sinus lies to the mesial side of the infundibulum.

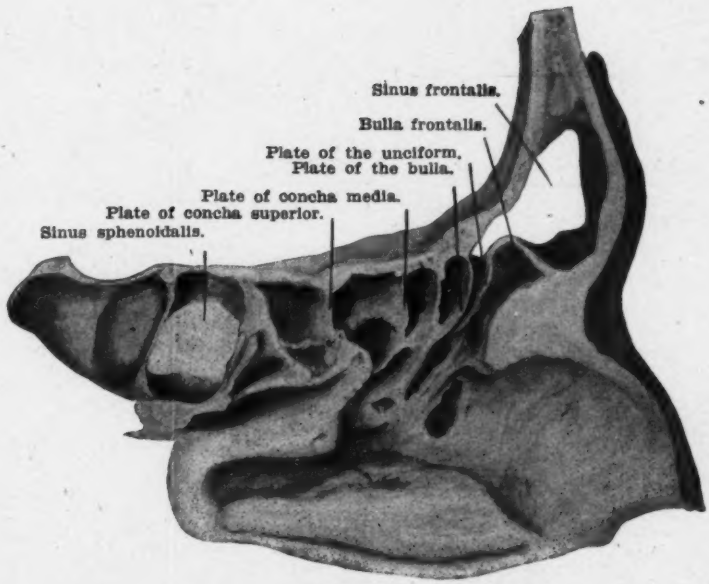

Fig. VI.

Sagittal section showing left side. The anterior half of the middle turbinated body has been removed and the ethmoid labyrinth has been opened up; the preparation shows an ethmoid cell which has developed below the unciform plate, pushing this up into the frontal sinus, where a bulla frontalis is formed; the other plates of the ethmoid are formed as usual.

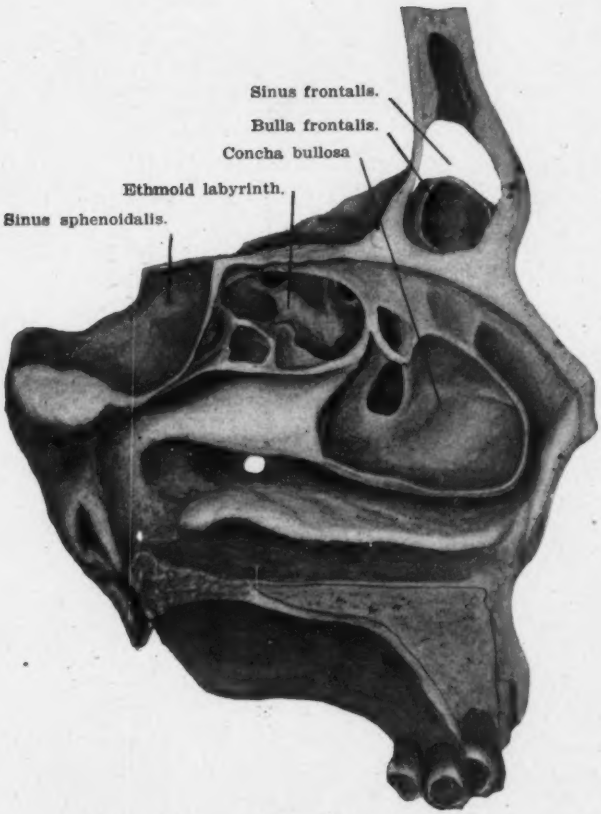

Fig. VII.

Sagittal section showing left side. The ethmoid labyrinth has been opened up; a large ethmoid cell is formed in the floor of the frontal sinus and an exceptionally large cell has developed in the anterior half of the concha media, forming the condition known as concha bullosa, also referred to as cystic enlargement of the middle turbinated body.

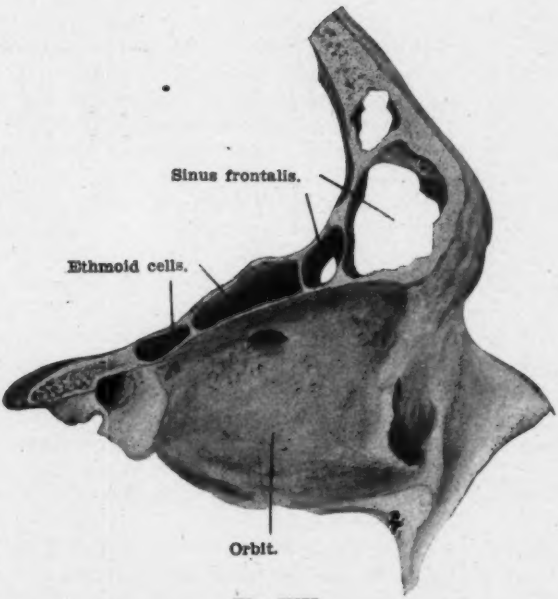

Fig. VIII.

Frontal section through the right orbit, showing sinus and ethmoid cells which have developed out over the orbit.

LII.

IS THERE AN IDEAL OPERATION FOR THE CORRECTION OF DEVIATIONS OF THE NASAL SEPTUM?

By Joseph S. Gibb, M. D.,

Philadelphia, Pa.

Ever since rhinology has taken its place among the special lines of study, septal deformities have been of all-absorbing interest. That the subject is not of easy solution is manifest from the innumerable operative procedures suggested for the correction of the same—procedures which are along divers lines.

A casual glance at the long list of measures suggested and the description of the defects to be corrected, convince one of the utter lack of unanimity of opinion.

Almost every author views the septum and its displacements from a different standpoint, hence their descriptions vary. To one unfamiliar with rhinologic lore, it would be difficult to recognize in any two descriptive operative measures designed to correct the same deformity much similarity in the preliminary description of the anatomic or rather pathologic position of the parts. For example, the description of the common forms of deviation as portrayed by Killian would scarcely be recognized as the same as those of Freer. Hajek looks at a septal deformity very differently from Krieg or Boennighaus.

What does such a lack of uniformity indicate? Surely not a want of accurate knowledge on the part of these men. They are all careful, conscientious, painstaking and patient workers in the field of research. Rather does it show that the form the septum may assume because of interference with the proper and normal development, or as a result of traumatism is so varied as to baffle accurate description. Doubtless each observer sees just the condition he describes, and just as truly no one sees them all. While it would be a matter of incalculable value to have a description which, while simple, would cover every possible shape and form the septum may assume, because of conditions which prevent normal development or distort it after it has been so formed, still the practical point, which is too frequently overlooked, is to determine in what

manner the distorted septum interferes with the proper function of the nasal chambers. It matters little from a practical standpoint whether the deviation is bow-shaped, S shaped, horizontal or vertical.

It is, however, a matter of much importance whether or not the deviation is in such a position or of such shape as to interfere with nasal respiration, or to obstruct the outlet from the various sinuses, or exert pressure upon sensitive nasal nerves. As indicated above, no one description can possibly serve as a guide for the study of septal deformities. The modern rhinologist must learn that each case is a law unto itself, and must be carefully and closely studied and if operative interference is demanded at all, that one selected which will give the most reasonable prospect of restoring, not the septum, but the nasal chambers to their normal functions. In order to accomplish this it is quite as important, perhaps even more so, that the posterior as well as the anterior portions of the chamber be the subject of investigation. Not infrequently we find marked deviation of the bony structures, either of the perpendicular plate of the ethmoid or of the vomer, even to the point of complete nasal obstruction, while the cartilage remains little if any out of its normal position.

It would seem in these days, when rhinologists have gone operation mad, an unnecessary refinement to point out or caution against the possibility of overlooking deformity in this body. Rather would we subserve the best interests of our art by uttering a word of caution against indiscriminate and unnecessary operation.

In this particular the writer is in entire accord with those who sound a note of warning. Swain has in graphic language refreshed our minds as to the real functions of the nasal chambers and given a word of warning as to the danger of our becoming nasal carpenters.

It seems to the writer that, in the nasal chambers as in other portions of the human frame, the point to determine is whether or not the malformation interferes with the function of the part, and if it does, what operation is best calculated to overcome the difficulty? If we have conscientiously settled this point we have gone a long way towards doing our patient a service.

In reviewing the operations which have been from time to time advocated to correct deviations of the septum, one is struck with the varied objects these operations have in view.

The early operations, e. g., the stellate punch, Robert's pin, Harrison Allen's supralabial and others, had but a single object; namely, to bring into a straight line a cartilaginous septum which from causes known or unknown had deviated from this position. With the possible exception of Allen's operation these methods took no thought of the bony septum or considered the presence of redundant tissue the result of inflammatory action. At the period these operations were in vogue little attention was given to the deeper nasal tissues, enough was accomplished it was thought, when the cartilaginous septum was brought into a position near the median line.

Later operations, designed by Asch, Watson, Gleason and others of the same general type, sought in addition to correcting the position of the cartilage also to make an effort to obtain the same result with the bony structures, which were known to have suffered as much as those more anterior. Also these operators saw the necessity in order to secure substantial results to take into consideration the redundant cartilaginous and bony tissues which filled in the concavity of a deviation or surrounded the site of a fracture, usually situated at the junction of the cartilaginous and bony framework. An effort was made to dispose of this redundancy of tissue in such a way that it would not obstruct the respiratory channel.

In all these earlier methods the desire of the surgeon was to effect a readjustment of the displaced parts with as little destruction or removal of the normal tissues as possible.

The object was much the same as the general surgeon has in view who endeavors to correct vicious union in a long bone by refracturing it and replacing the fragments in as nearly a normal position as is possible to get them.

The modern operation—the submucous resection—as practised by its many advocates,—notably Killian, Hajek, Krieg and Boennighaus in Europe, and Freer, Ballenger and White in America—is designed upon lines totally different from all former septal operations. This method sees in the distorted tissue, be it cartilage, bone or adventitious product, the result of inflammatory action—an obstruction to be removed—and if it interfere with nasal function is resected from between the layers of the overlying mucous membrane.

In this rapid and cursory review of the salient features of the operations designed for the correction of deviated septa, it will be seen that all the older methods sought to preserve intact the septum.

The modern operation—the submucous resection—has no such object in view. The septum is regarded as the offending body and no effort is made to preserve it. Claims are made that from an anatomic or physiologic standpoint this tissue is unimportant, and hence no harm will result from its ablation. Therefore, if it is necessary in order to secure a free and unobstructed naris, the cartilage or bone be removed, this is done without hesitation.

With a view of ascertaining the advantages of the submucous operation, the writer began the use of this method about two years ago.

Previous to this period each case was studied in its entirety and that operative procedure employed which seemed to offer the best opportunity of restoring the nasal chambers to their normal functions.

The desire in view was to accomplish this result with as little interference with the structures as was possible. In cases in which the removal of an enchondrosis or exostosis gave promise of obtaining sufficient space to secure good nasal respiration or relieve pressure, this simple operation was done, and the septum undisturbed.

If after a careful study of the case it did not seem possible to secure the result by the simple manner detailed, then one or other of the various operative procedures upon the septum in vogue at the time were employed.

In the eariler years the Robert's or Adams' operation and later the Asch, Watson, Gleason, or those of the same type, were among those selected.

While complete success did not always crown our efforts still there was a fair measure of it.

In a paper* upon this subject read at the American Medical Association, in June, 1904, the writer selected 100 cases and tabulated the results as follows:

Total number of Asch operations 65
Total number of Watson-Gleason operations.....23
Total number of spurs removed operations.....12

Of the Asch operations:

The result was good in 47

*Gibb, J. S. Deviation of the Nasal Septum. A review of 100 operations for correction. Trans. Sec. Laryngol., Am. Med. Assn., 1904.

The result was fair in 11
The result was failure in..... 3
The result unknown in 3

In one case of failure by the Asch a successful result was obtained later by the Watson-Gleason.

Of the 23 Watson-Gleason operations:

The result was good in 19
The result was fair in 3
The result was failure in 0
The result unknown 1

There were twelve cases in which it was deemed sufficient to remove only an exostosis or enchondrosis, and of these all were successful.

It was not claimed that a perfectly artistic result was secured, that the septum was made to follow an absolutely straight line, but what seemed to the writer more important in those cases marked with good result, the respiratory function of the nasal chamber was restored or pressure removed or both.

In passing, it is to be noted that of the 47 Asch operations nearly all were plain cartilaginous deviations, while the Watson-Gleason were combined osseo-cartilaginous.

Since the writer first began the use of the submucous resection method, he has employed it in 25 cases—not a large number, to be sure, but sufficient to form a basis for comparison. In many respects this method is immeasurably superior to any operation which has yet been devised for the correction of deviated septa; that it is a perfectly ideal operation and adapted to every septal deformity the writer does not admit. This method far surpasses any of the older ones in the simplicity of the after-treatment. Again it is superior for the reason that when skillfully performed it ensures a perfectly straight septum, one that cannot possibly revert to its former faulty position. These are weighty arguments in its favor.

However, there are equally strong arguments against the universal application of this method. (1) It is an operation difficult to execute and success will result only in skillful hands. (2) It requires for its accomplishment a much longer time than is necessary for any of the older methods. (3) Which is dependent upon the (2), it is a severe strain upon the endurance of both patient and surgeon. None of these criticisms are offered to disparage the operation, but merely to place them side by side with less radical procedures in those

cases in which it might be thought feasible to employ mild measures.

The writer disclaims any spirit of harsh criticism of this method, and he doubts that serious consideration would be given him if he did, since praises are raised to this new method in every medical journal, book and society in the land. The operation has come to stay and is an immense advance in our knowledge of septal surgery. It is believed, however, wise to sound a note of warning against the indiscriminate use to the exclusion of all other and well tried plans of septal improvement.

In skillful, judicious hands it will prove a great boon to suffering humanity. In unskillful, bungling ones it will be a curse.

Already cases are coming to our notice where this operation has been attempted which would have been better had no operation been performed.

Teachers assume a grave responsibility when they recommend this operation to their classes without at least a word of warning as to the difficulties, and perhaps coupled with the advice to the tyro to first perfect himself in the technic upon the cadaver or lower animals.

From the fact that from time to time new operations have been designed to correct septal deformities, it may be well argued that previous attempts had not given universal satisfaction. As with incurable diseases the number of remedies are legion, so with deformities of the septum surgeons have not been satisfied with the efforts of their predecessors, and have ever striven to design a method that would be ideal—hence the number of operations have multiplied in the effort to attain this goal. Has this Utopia been reached? The advocates of the submucous resection method believe that it has and that every conceivable form of septal malposition may be corrected. Doubtless in extra-skillful hands this statement is strictly true—but it is only a partial truth as to the claim that the operation is ideal. Granted that in the hands of a master, a deviation may be dissected out from its encompassing mucous membrane, it may not always be the part of wisdom to so dissect it. For example: It is no uncommon experience to find a septum deviated only in its posterior bony portions. The perpendicular plate of the ethmoid, the vomer and the maxillary crest, each or all participating in the deformity,—and the

latter surrounded by new tissue the product of inflammatory action, either cartilage or bone according as one or other of these tissues is the site of the initial inflammatory process, the whole forming a mass which entirely prevents free nasal respiration. Given such a case, is it the part of wisdom to remove a perfectly normal tissue, one that is in nowise productive of harm, in order that an offending, obstructing mass may be reached and removed? This question could well be answered in the affirmative if there were no other way,—but that this is not the case is attested by the army of rhinologists who have successfully laid siege to and conquered these conditions long before the advent of the submucous method. Force, again, is added to the argument against the wanton destruction of normal tissue, from the fact that all the advocates of the new method admit the exceeding difficulty of stripping the mucoperiosteum in the posterior deviations. So peerless an operator as Freer* advises the operator to persevere with the resection of the bone at this point and not to allow the fear of a tear of the mucous membrane or a perforation to cause him to desist in his efforts. The older methods could do no more injury than tear the mucous membrane, or cause a perforation, and in the latter method the septum would remain intact—a very important consideration notwithstanding the assertion that the cartilaginous septum plays but a feeble role in sustaining the tissues of the bridge.

Of course in those cases in which the bony deviation is associated with a marked cartilaginous bend, these criticisms would not be relevant, since the one may not be satisfactorily corrected without the other, and it would seem wiser to allow one operation to suffice for the entire deformity. But even in this latter, in consequence of the extreme difficulty in effecting thorough stripping of the periosteum from the bone, the embryo or unskillful rhinologist had better select some less difficult, though perhaps also less efficient, method until he has acquired sufficient skill to make a successful result measurably sure.

Nor is this particular type of deviation the only one in which the submucous resection operation appears an unnecessarily harsh one. A very common deviation is a bowed cartilaginous septum with no change in the bony structures.

It is at once admitted that these are readily corrected by the submucous operation, and that they are comparatively easy

*Freer. Annal. Otol., Laryn. and Rhin., June, 1905, page 242.

of execution. But it is also just in these cases that any one of several of the older methods of operating have scored signal successes.

It is believed that the poor results of the older methods which seemed to necessitate the application of newer and better means arose from a lack of appreciation of the influence of the bony structures in reproducing deformities which were believed to be thoroughly corrected.

Further, the writer believes that were deviations confined alone to the cartilaginous septum, the older methods would have sufficed and there would be no need of the submucous resection.

In this type then, while it is conceded the submucous operation gives excellent results, it seems to the writer that at least in properly selected cases, it is an act of folly to discard those methods which have served us so well in the past—and that it is better surgery to save the cartilage and effect by simple means the results we desire.

Shall we then abandon all our old methods for the correction of septal deformities and adopt only the new? Have we an ideal operation?

If we are correct in our premises that the subject of septal deformities is so complex as to baffle accurate description as to the conditions to be overcome, manifestly no one operation can be relied upon to correct them. For a quarter of a century measures have been employed with more or less success. We should, with this experience, have been taught lessons which should enable us to determine in what class of cases success might be looked for, and those in which success seems improbable, adhering to the old methods in the former and relegating the latter to the newer.

An effort has been made to point out a few conditions in which it is believed as good success may be attained, with less destruction of tissue, by the older methods. There are many others. In view of the multiplicity of abnormal shapes and positions a deformed septum may assume, each case must be a study in itself, and the surgeon select that operative procedure which his experience and individual judgment has taught him is best suited for that particular case. While it is admitted that many, if not all, deviations may be corrected by the submucous resection, it is denied that it is a good surgical procedure to resort to it in every case.

Therefore it is believed to be unwise to abandon all the

labors of our predecessors, many of which have stood us in good stead in the past and take up with new. Rather does it appear more judicious to welcome the new as a great addition to our means of attacking this difficult and intricate subject, helping us out of many difficulties in which we were formerly helpless, but at the same time holding fast to the old well-tried and satisfactory methods.

The writer trusts he has been able to make himself perfectly clear on the points at issue. He disclaims any desire to disparage or attack the submucous operation. On the contrary, he has been filled with admiration of the conception of this operation and of the results obtained in the hands of the little band of expert operators throughout the world who have enthusiastically advocated its claims. The thought is that harm will surely result from an indiscriminate endorsement of this truly brilliant procedure unless an attempt is made to give it the position it deserves in the surgery of the nose. The writer is conscious of the fact that the attempt to so place it is feeble, but he also believes that ultimately and in abler hands it will be so placed and result in inestimable benefit to mankind.

From what has been said we are in a position to answer the question—is there an ideal operation for the correction of deviated septa? And it must be in the negative.

The conclusions reached are:

1. There is no single operation devised which will correct every form of deviation of the nasal septum.

2. The shape and position of the septum resulting from deviation is so various as to make accurate description difficult, if not impossible.

3. The object of operation upon the septum should be correction sufficient to restore the normal functions of the nose with as little destruction of tissue as possible.

4. Each case must be a law unto itself and studied carefully and in its entirety.

5. In many cases the object sought, namely, restoration of nasal function, may be attained by the removal of exostoses or enchondroses and the septum left intact.

6. Other cases may be corrected by one or other of the older septal operations, the selection of which depend upon the individual preferences of the operator.

7. Still others can be best corrected by removing the septum entire according to the submucous method.

LIII.

PATHOLOGY OF THE FAUCIAL TONSIL.

By James A. Babbitt, M. D.,

Philadelphia.

The assignment of a topic upon the pathologic conditions of the tonsil presents a field most comprehensive in its scope, but its fortunate limitation to a surgical symposium in certain degree diminishes this range. While the tonsil is comparatively simple in histologic structure, its important position at the alimentary inlet, close relation to cervical lymphatics, remarkably free vascularity, and exposure to constant impact from foreign material, render it anatomically and functionally of considerable significance.

These minor groups of adenoid tissue comprising the so-called adenoid ring, omnipresent and familiar almost to a degree of contempt, save in relation to this anatomic interference and retardation of functional development, still in large measure defy scientific investigation as to their exact import in the body economy, and still leave indiscriminate surgical interference a question of justifiable debate.

Most admirable and painstaking work has been done upon this investigation by Packard, Wood, Swain, Wright, Browne, Hall, Tilly, Kyle, Allen and others, yet notwithstanding their splendid contributions to this field of literature, the tonsillar function still remains a matter of opinion.

In view of noteworthy studies already published on cryptal degeneration, lymphogenetic function, tubercular significance, focal areas for bacteria, vasomotor connection, etc., it would seem best in this paper, after brief outline description of anatomic and physiologic characteristics, broadly to classify and describe pathologic conditions present, and finally, perhaps, discuss in more detail some special topic of practical application in the routine surgery of this part. Inasmuch as chronic hypertrophy and its resultant effects are most significant upon general metabolic activity, the progressive change of the tonsil during the developmental period would

perhaps be primary in importance, and next in order the systemic effects of cryptal absorption.

SUGGESTED ORDER OF STUDY.

1. Brief review of anatomic and physiologic characteristics.
2. Classified description of pathologic conditions acute and chronic.
3. General effect of hypertrophic enlargement in the naso-pharyngeal area.
4. Correlated physiologic influence upon general metabolism.
5. A comparative study of hypertrophic changes at stated age intervals.

Anatomically, the faucial tonsils, lying just internal to the angle of the jaw, between the palatoglossus and palato-pharyngeus muscles, consist of a mass of glandular tissue, flattened ovoid in shape, of the type of compound lymphatic glands, and in early life about three-quarters of an inch in general dimension. Each tonsil contains from twelve to fifteen cryptal orifices, formed by invagination of the epithelial layers of mucous membrane, and from these crypts numerous irregular communicating channels branch out into the subjacent parenchyma. In the walls surrounding the crypts are found numerous nodules, without, however, directly communicating ducts, the contents being discharged directly through the epithelial walls of the crypt. The pharyngeal surface of the tonsil is indurated from constant foreign impact, and the crypt mouths become partially occluded by cicatricial bands of fibrous tissue in later life. The mass is closely adherent to the superior pharyngeal constrictor, often adherent to anterior and posterior pillars. Harrison Allen has called attention to the almost uniform presence of a sulcus dividing the lower smooth from the upper cryptose position and a posterior velar portion. According to Piersol, the epithelium lining of the crypts becomes completely infiltrated with lymphatic cells.

Beneath the indurated cortex, a much softer parenchymatous portion exists, and closer study of lymphoid follicles shows analogy to Peyer's patches and solitary glands of the intestine. Within the crypts the epithelium shows marked degenerative changes, and in adult life small cheezy pellets are often present, formed from accumulated secretion, and from time to time ejected.

Attention should be called to the supratonsillar fossa upon

and back of the tonsil covered by the plica triangularis, inasmuch as this is a most important pathologic focus.

The vascular supply is derived from the tonsillar, lingual, faucial, internal maxillary and pharyngeal arteries—and the nerve control by branches from the trifacial and glossopharyngeal.

Of important significance is the lymphatic outlet into upper deep cervical nodes, lying along the internal jugular vein, and thus communicating with the axillary and superior mediastinal spaces. Its embryologic development has been most diligently studied by many authors, but is without the province of this paper.

In anatomic summary, then, tonsillar points of interest are:

1. Mucous membrane and crypts.
2. Interfollicular tissue.
3. Lymphoid follicles and nodules.
4. Fibrous tissue stroma.
5. Vascular supply.

Physiologically the tonsil presents a far more difficult problem as the data available on functional importance are meagre, and many authors merely consider these glands adjuvants to salivary work in deglutition. Wood, who has carefully covered this field, emphasizes the lymphogenetic possibilities; others the phagocytic, absorptive, etc.

Inasmuch as the tonsils contain closed lymphoid follicles, a first inference would be that they should be included in this category of the socalled ductless glands and be estimated with the thyroid, suprarenal and others. Indeed, their embryologic relations with the second branchial cleft would strengthen this assumption. Yet a study of the secretion fails to reveal anything commensurate with the renal, colloidal and other associated fluids. Its contribution to the saliva and lymphatic manufacture, partly chemical and partly physical in importance, probably limits its field in this direction.

The fact that toxemic and other materials are frequently absorbed through the crypts would bring next consideration to its possible share in alimentary absorption and nutrition. Its connection with the lymphatic channels is certain, and this possibility is not remote, yet its surface is so comparatively small and extirpation shows so little disturbance that it seems almost inconsequential.

It has been suggested that the tonsils through moderate

vasomotor reaction may serve some part in protecting the pharyngeal cavity by influencing secretion. This, again, has no corroboration.

Inasmuch as the throat first receives pathogenic matter admitted to the system per oral cavity—its protective and possibly antitoxic properties should be considered. Epithelial tissues in general have a positive selective resistance to toxic absorption, and the constant formation of leucocytes would indicate phagocytic action. The alimentary mucous membrane, universally glandular in type, preserves this resistance in uniform degree, as per evidence of the immunity to ptomain contact in the intestines, and it is entirely possible that the free display of adenoid tissue in and about the pharyngeal cavity, and particularly in the tonsil with its specialized tissue formation, may contribute to this capability of resistance.

Newcomb has called attention to the constant rarefaction in the epithelial surface of the crypts as the tonsil atrophies, allowing various organisms to pass through the thin walls, again strengthening the theory of protective function during the period when all adenoid tissue is most marked.

In experiments upon colors rubbed into the hypertrophic tonsils, Goodale found them taken into the interfollicular space but not into the follicles; and attention has been called to the free phagocytic migration from the center to tonsil surface functionally protective in destroying septic organisms so prevalent in the mouth.

2. CLASSIFIED DESCRIPTIONS OF PATHOLOGIC CONDITIONS, ACUTE AND CHRONIC.

The variance in terminology renders a definite classification of tonsillar affections somewhat difficult. By many authors the term lacunar has been substituted for the appellation "follicular" customarily applied to such common condition of the tonsils, as distinguishing the cryptal inflammation from that involving lymphoid follicles.

A simple classification based as much on symptomatology as microscopic change might be as follows:

(a) *Acute.*

Inflammatory, superficial or catarrhal tonsillitis.
Lacunar or cryptic tonsillitis.
Peritonsillar abscess.
Acute ulcerative tonsillitis (rare).

Membranous tonsillitis.
Herpetic tonsillitis.

(b) *Chronic.*

Chronic hypertrophy of the tonsils.
(*a*) Fibroid.
(*b*) Soft adenoid enlargement.
Chronic lacunar tonsillitis.
Mycotic inflammation of the tonsils.
Tonsillar calculi and foreign bodies.
Traumatic and escharotic affections.
Rheumatoid tonsillitis.

II. *Neoplasms.*

(1) Benign tumors of the tonsils.
Papillomata.
Fibromata.
Angiomata.
(2) Malignant tumors.
Carcinomata.
Sarcomata.

In addition, cases of lipomata and lympho-sarcomata have been reported.

In all these various degrees of acute and chronic inflammation of the tonsils, the bacterial germs most frequently involved are the pyogenic cocci, but these will be considered in the discussion of inflammations.

Acute Types.—1. In acute catarrhal or superficial tonsillitis, as termed by Kyle, the inflammatory condition is that of a simple glandular mucous surface. First, distention of blood vessels; second, exudation of fluid and diapedesis of cellular elements from central current, and third, various degenerative changes in proximal fixed tissues and proliferation of tissue cells.

This simple inflammatory condition, with customary symptomatic changes of increased temperature, redness, thickening and modified nutrition, may undergo resolution or advance into more defined lacunar types, producing surface necrosis or involving deeper reticulated and lymphoidal structures of the tonsil.

In these inflammatory stages we note the marked phagocytic effort of the leucocytes to resist pathogenic microorganisms, by massing themselves about, absorbing and digesting toxic elements.

2. Acute lacunar (often termed follicular tonsillitis), like

the catarrhal form in advanced stage, is characterized by considerable accumulation of inflammatory exudate in the crypt passages. This includes epithelial cells, leucocytes, bacteria, and various forms of debris, and in the chronic stages, masses of keratoid material formed by epithelial degeneration. Associated with this will be found a marked degree of cell proliferation in adjacent tissues, and inflammatory exudate into the parenchyma, producing infiltration of leucocytes and an edematous condition of adjacent glands.

Paterson and Sawtelle demonstrated that the supratonsillar fossa which has almost direct connection with superior crypts and follicles, was the seat of primary entrance for tonsillar infection.

Microorganisms' presence in the tonsils have been investigated by many authors.

Streptococci, both forms, staphylococci (aureus and albus) and the diplococcus predominate; in lesser frequency the pneumococcus, Klebs-Löffler bacillus, staphylococcus citreus, micrococcus tetragenus, micrococcus albus liquifaciens, leptothrix fungus and others more sporadically. The essential qualifications for most bacterial growth are here present in the deeply lying crypts—warmth, moisture and nutritive pabulum, augmented by inflammatory change.

It has been shown by recent research that the tonsil is often the seat of primary and secondary tubercular deposit, while in general rheumatic fever and the exanthematous diseases, the tonsillar condition is primary in many cases.

Allusion might here be made also to the specific infection of syphilis and its attendant chain of ulcerative and necrotic conditions.

3. Parenchymatous tonsillitis. Beyond the cryptal structure of the tonsil we find secondary isolated nodes with an inner follicular parenchyma, and this divided by fibrous trabeculae passing in from the capsule and carrying vessels. Associated with lacunar inflammatory change, this entire parenchymatous mass may become involved with characteristic exudate. This may readily resolve in time to tonsillar abscess with deep encysted pockets.

4. Peritonsillar abscess is closely allied to the general parenchymatous inflammation, in fact abscess of true tonsillar tissue most often commences in the peritonsillar area.

According to Shurley, the first step in peritonsillitis is de-

termination of the blood to the tissues caused by toxic action on blood vessels. In fact, anything seriously disturbing relations between internal and external capillary circulation may be an exciting cause.

Peritonsillar abscess or quinsy is so familiar to specialists and general practitioners as to require little description. Its process is essentially inflammatory, involving surrounding pharyngeal and lymphatic tissues; a rapid infiltration and cell proliferation, marked effusion from vessels and lymphatic channels, leucocytes subject to chemotaxic attraction. Necrosis takes place and abscess points in line of least resistance, spreading freely if in outer thin tissues, but often requiring deep incision through body of tonsil when confined to tonsillar structure proper. The tonsillar cyst has also been described in this connection.

5. Acute ulcerative tonsillitis per se is infrequent and is closely allied to necrotic areas secondary to aphthous deposit and the small ulcerations left upon removal of herpetic blebs.

6. The membranous or aphthous is very similar to membrane produced by the Klebs-Löffler bacillus. The membranous deposit is continuous with that in the crypts, and may spread over the entire tonsil and neighboring pillars. It is in form a coagulation necrosis, producing surface ulceration of varying depth.

Superficial layers of mucous membrane are infiltrated with pus cells, exudate and bacteria. It consists of grayish membrane, often bleeding if detached, and often an exanthematous complication. As has been noted, non-pathogenic bacteria become pathogenic under such conditions of weakned tonsillar resistance.

7. Herpetic tonsillitis was early described by Moure of Bordeaux as consisting of small vesicles which break in twelve to twenty-four hours, leaving ulcers covered with whitish exudation. It is likewise a coagulation necrosis of surface epithelium with vascular exudate.

The tonsillitis due to rheumatic diathesis is so closely allied to the catarrhal and parenchymatous types that we have not here given it special classification.

1. *Chronic Types.* The omnipresent chronic hypertrophy of the tonsils may be either the true hyperplasia of all tonsillar structures or a hypertrophy of the connective tissue elements. It is an almost invariable sequence to repeated attacks of acute

tonsillitis and probably has been most often investigated of all pathologic conditions.

The appearance of the hypertrophied tonsil is characteristic. A large lobulated mass projecting well beyond the margins of the pillars with crypts freely visible, contracted or open in accordance with the age and progression of hypertrophy.

Lennox-Browne classifies these forms of hypertrophy as 1. Peninsulated or projective. 2. Cowled. 3. Lacunar, honeycombed or ragged, and in morbid anatomy as—1. Interstitial or sclerotic. 2. Lymphoid.

Bosworth thinks one-third to one-half of this number of cases arise from infectious diseases and places the etiologic order: first, scarlet fever; second, diphtheria; third, measles; fourth, smallpox. Unilateral hypertrophy has frequently influenced suspicion of syphilitic origin or development of neoplasm.

Unquestionably the diathetic or strumous diathesis and the exanthematous diseases are in large part causative of this condition. Suchannek claims that infection most often begins in Waldeyer ring, especially the faucial tonsil, and Packard reports five cases of endocarditis all preceded by attacks of amygdalitis.

According to Price-Browne, tonsillar hypertrophies are due to: 1, congenital causes; 2, diathetic conditions; 3, exanthematous diseases; 4, congenital syphilis, rheumatism, etc.

Its actual pathologic change consists, as stated, either in general hyperplasia of all glandular elements with cell proliferation and infiltration, or marked overgrowth of fibrous tissue, trabeculae and subsequent contraction. Its associated pathologic influence will be discussed later.

2. *Chronic Lacunar Tonsillitis.* This follows repeated attacks of the lacunar type, and inasmuch as the crypts are often partially obliterated by fine fibrous tissue trabeculae, caseous plugs of inspissated secretion (often keratoid mass) commingled with decayed food products and possibly bacteria, are found in crypt pockets. Often these are exuded as firm, almost osseous masses. (Wood claims a non-pathogenic state here.)

3. Mycotic inflammation is due to a fungous mass, essentially a form of necrosis, and usually arising from the crypt walls, though often spreading to the surrounding surface and even pillars. The leptothrix bacillus is usually causal and the condition is comparatively benign.

4 and 5. Calculi, foreign bodies, traumatic abrasion,

escharotic injury, etc., produce pathologic changes of acute and chronic inflammation respectively, and in this category might possibly be included drugs influencing inflammatory changes, such as mercury, antimony, potassium iodid, arsenic, copper, lead, zinc and belladonna.

In reference to calculi it has been maintained that the supratonsillar fossa is an especially vulnerable position for such concretions, that these vary from small specks even to an ounce weight, and resemble tartar on the teeth.

Gruening states that all tonsillar concretions contain leptothrix and that this influences precipitation of lime in form of chalk, while others hold that tonsillar calculi consist principally of phosphate and carbonate of lime.

Neoplasms have been especally well studied by Browne, Hall and Tilley and the older Continental investigators. It is sufficient here to allude to the main characteristics which resemble similar tumors in other glandular regions. Of the benign tumors the papillomata are wart-like excrescences emerging from the crypt walls or found at the supratonsillar portal. The fibromata are usually pedunculated or sessile, polypoid in appearance and likewise protrude from the crypt margin or adjacent tonsillar surface. In the malignant group carcinomata are of the epithelioma type, and may be squamous, columnar or alveolar, while sarcomata may be of simple type, such as round or spindle celled, and compound, as the angiosarcoma, lipoma and myxosarcoma (lymphosarcomata have also been reported).

The tonsils as portals of infection are elsewhere discussed in detail.

3. GENERAL PATHOLOGIC EFFECT OF HYPERTROPHIC ENLARGEMENT IN THE NASOPHARYNGEAL AREA.

The pathologic effect of chronic hypertrophy is almost directly that of respiratory interference.

Primary objective symptoms are localized functional obstruction, a muffled, nasal or cracked voice, difficult articulation, embarrassed deglutition, frequently a chronic hacking cough, high narrow palate arch, constricted nares, drooping lower maxilla with faulty dental alignment, vitiated sense of taste, smell and hearing, dull and listless facies, stupid expression and thickened lips, mouth-breathing and oral membrane impaired by exposure.

Of deeper and secondary significance are chest deformities,

such as pigeon or chicken breast, disturbed night rest with heavy snoring respiration, habit choreas and stuttering, fetid breath, increased susceptibility to bacterial invasion, increased tendency to contract bronchial affections, and general systemic malnutrition.

These and many other primarily functional and secondarily organic deficiencies, really pathologic in import, can be traced directly to the hypertrophied faucial tonsil.

4. CORRELATED PHYSIOLOGIC INFLUENCE UPON GENERAL METABOLISM.

Next should be considered the physiologic influence of this hypertrophy upon general metabolism.

Much of the foregoing enumeration rightly belongs to the category of this heading, but in addition emphasis should be placed on these points:

1. Direct relation of the tonsils to deep cervical nodes and general lymphatic system.

2. Remarkable vascularity in tonsillar mucous cortex and subjacent parenchyma.

3. Close relation to cervical sympathetics and consequent systemic influence of chronic irritation.

By reason of free vascularity, no nasopharyngeal area more readily responds to both local and distal venous engorgement, and consequent stasis and diapedesis produce secondary ulcerative and parenchymatous inflammation in quick sequence.

Through the cervical nodes directly, and through lymphatic channels, salivary glands, ethmoid and sphenoidal sinuses more remotely, even the axillary spaces and pulmonary orifices are vulnerable.

Close relation to both cerebrospinal and sympathetic nerve systems afford means for low grade neurotic irritation in the chronic inflammatory forms, and favor the severe algesia so distinctive of tonsillar and particularly peritonsillar involvement.

Again, a further important element in the consideration of tonsillar pathology, is the favorable focus the faucial tonsil presents for aiding the absorption of the many products of intermediate metabolic change in the albuminoid metamorphosis.

Granting the possibilities of salivary absorption, and also the dangerous probability of cryptic stasis and accumulation of semitoxic bacterial products, these degenerative changes may have a potent influence in producing the lethargic con-

ditions of the body attendant upon tardy elimination. Absorption of toxins accidental or experimental has proven moderately rapid. Granting again the more or less constant presence in tissue interstices, fibrous sheaths and recticular spaces of products of intermediate metabolic transition, etc., and for a limited space remarkable exposure in this area, why should not this metabolic influence be consequential?

It is difficult to locate the cause of discomforting growing pains of early childhood, the low grade lethargic states of systemic function, frequent states of mental dullness and apathy, dull mottled skin. Is it not plausible to assume that free absorption of toxins through degenerated cryptal contents may have at least some influence in producing and maintaining these retrograde katabolic changes?

5. COMPARATIVE CHANGES IN CHRONIC HYPERTROPHY.

It has been the writer's effort in outpatient service at the Children's Hospital, to study comparatively and statistically the systemic and organic alterations accompanying this most common malady. Unfortunately at the present time sufficient data of value have not been obtained for statistical report.

The relief from pharyngeal obstruction when such appears pathogenic, is marked and almost constant, but are there no fundamental grounds for judging hypertrophy and chronic lacunar tonsillar conditions, as essentially abnormal?

In consideration of this must be borne in mind the ready tendency to abrasion when hypertrophied, and consequent rapid absorption.

In connection with the paper the writer has been making comparative investigation of hypertrophic tonsillar changes at different age intervals. (This in conjunction with Dr. Scarlett, of the University of Pennsylvania.) Its purpose has been to ascertain if possible by sufficient ultimate averages the distinctive changes, particularly in the parenchyma of the tonsil. Data obtained, however, have not in average presented sufficient distinctive characteristics to warrant judgment. A number of slides have been obtained covering the marginal periods of normal adolescent growth, and a subsequent report will be forthcoming.

In general summary upon the all-important topic of tonsillar hypertrophy the following tentative conclusions will be accepted:

a. The tonsils become permanently hypertrophied as a result of single or repeated acute inflammatory changes.

b. These changes are both hyperplastic and hypertrophic in character.

c. The hypertrophy is most common between the ages of four and twelve, disappearing in atrophic change between thirty and thirty-five years of age.

d. The lymphoidal action gradually retrogrades upon occlusion of the eliminating surfaces and ducts.

e. Systemic absorption may be augmented by the atrophic thinning of the epithelial lining and decreased leucocytosis may accompany abrasion of the surface.

i. Whatever of functional importance may have attached to enlarged tonsils during periods of special lymphoidal activity, in all probability does not exist when such period has ceased.

j. Statistics do not indicate any systemic deprivation either general or in special sense organs after indicated surgical terference properly indicated.

LIV.

ATROPHIC RHINITIS.

(RHINITIS ATROPHICANS SIMPLEX ET FOETIDA, SEU OZAENA.)

By Dr. John Sendziak,

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EARLY HISTORY.

The relation between atrophic rhinitis and so-called ozena as well as their essential character has not up to the present been wholly elucidated.

The term ozena is found in medicine since the earliest times. Primarily, the name "ozena" was given to all pathologic processes of the nose associated with exhalation of a disagreeable odor. Later, however, it was limited to fetid ulceration of the nose (Pliny, Celsus). Galen distinguishes two kinds of ozena: (1) Simple ulcerations difficult to heal. (2) Those associated with a disagreeable odor.

Paulus Aegina regarded ozena as rottening and purulent ulceration, caused by saturation of the interior of the nose with sharp humors. This opinion, of which Aetzius and Trallianus were also adherents, was maintained until the twelfth century. Actuarius first proved that an ulcerative process is not necessarily present, and that the ozena is caused by a decomposition of the secretion, although some authors, Ambrose Paré, for instance, held to Galen's opinion.

This rational theory of Actuarius was further developed at the beginning of the seventeenth century by Joannes Crato, who regarded ozena as an infectious disease, which latterly has been proved by Capart.

So great, however, was the authority of Galen then that such authors as Fabricius, Aquapendente and Sir Thomas Mayern returned to the opinion that ozena consists in ulceration of the nose, often associated with syphilis.

At the end of the seventeenth century Vieussens, Reininger and Günz maintained that ozena is not an independent disease, but depends upon suppuration from the nasal sinuses, which opinion has been warmly advocated of late by Grünwald of Monaco.

RECENT HISTORY.

Weber in the second half of the nineteenth century (1860), regarding ozena as not an independent pathologic process but as a symptom, was of the opinion that the term ozena is superfluous. At present such authors as Castex, Grünwald, Krakauer, etc., are of like view. Solis-Cohen proposes "coryza foetida" instead of this term. Seifert dispenses with it also, distinguishing only "rhinitis atrophicans simplex" and "rhinitis atrophicans foetida"; and he regards both forms as varieties of rhinitis chronica.

Likewise, Krieg, author of the excellent monograph in Heymann's Handbuch, distinguishes "rhinitis atrophicans crustans non-foetida" and "foetida seu ozaena," regarding both pathologic processes as one. With this opinion, however, the French school does not agree, especially Tissier, Gougenheim, Baratoux and Charazac. Moure distinguishes five forms of ozena: adenoid, sinusal, necrotic, purulent and atrophic. Finally Cozzolino proposes the term "Keratosis degenerativa saprophytica," and Sticker "xerosis," identifying it with tabes.

Since then and up to the present, confusion reigns both as to the term itself and the nature of the disease.

Upon no other disease in rhinology and laryngology, except perhaps diphtheria and tuberculosis, have so much time and paper been wasted as upon "atrophic rhinitis." It is enough to say that the papers touching this question, especially etiology and treatment, have reached an imposing number—about 500. Of these, the greater portion belong to America (105), Germany (98) and France (90). Other nations include England (35), Italy (23), Austria (14), Belgium (13), Spain (9), Switzerland (8), Holland (6), Russia (5), Denmark (5), Poland (4) and Sweden (1).

Of the best papers I must mention here, above all: In America, John Mackenzie's, of Baltimore (1884), Mulhall's, of St. Louis (1885), Wright's (1891), Wilson's (1892), Kyle's (1894—an excellent bacteriologic paper), Seiss's (1896), etc. In Germany, Höffler's (1885), Gerber's (1890), Grünwald's (1893), Hopmann's (1893), Abel's (1893—bacteriologic), Krieg's (1897—an excellent monograph), Cholewa's and Cordes (1898) and finally the very good monograph of Grosskopf (1902). In France, Loewenberg's (1884—bacteriologic, published at the same time in German), Lacoarret's (1888), Moure's (1888), Tissier's (1894), Lautmann's (1897).

In England, Williams's (1887), Haviland Hall's, etc. In Italy, Belfanti's and della Vedova's (1896—bacteriologic) and de Simoni's (1897). In Austria, Habermann's (1886) as well as Hajek's (1886). In Switzerland, Wyss's monograph (1886) and Siebenmann's (1900). In Poland, Baurowicz's (1895—bacteriologic).

DEFINITION OF ATROPHIC RHINITIS.

In accordance with the generally accepted opinion (B. Fraenkel, Baginsky, Gottstein, Beverly Robinson, Krieg, Grosskopf) by the term atrophic rhinitis, which I shall divide into rhinitis atrophicans simplex and foetida, regarding them as one pathologic process (Krieg), I understand chronic catarrh of the nose, not having an ulcerative or destructive character, which may spread to the pharynx or larynx, and which differs from simple chronic catarrh by a more or less thick, quickly drying secretion, leading to the formation of crusts which are characterized by a very strong, disagreeable, specific odor (ozena), and by atrophy of the mucous membrane and bones of the nose.*

PATHOLOGIC ANATOMY.

Under the microscope the mucous membrane shows a chronic inflammation, hence masses of leucocytes with fatty detritus are seen; the epithelium is changed from cylindrical to squamous (one or many layers) with cornification of the superficial strata (Valentin, Suchardt, Seifert). The glands, acinous and tubular, are atrophied and with them the olfactory apparatus. Later the inflammatory cells are changed more and more into fibres of connective tissue, and then the mucous membrane becomes cicatrized, thin, hard and dry. Atrophy of the bones of the turbinates ensues; they become thin, friable, sometimes perforated, undergo changes in form; the inferior border of the turbinates—normally convex—becomes straight or even concave finally. The nasal septum as well as the bones of the face also undergo the atrophic process (Zuckerkandl). The accessory sinuses of the nose are often affected, the mucous membrane is dirty gray, bleeding and covered with dirty green mucus or mucopurulent secretion (Hartman, Krause, Zuckerkandl, Haberman).

[*Part of this paper relating to the Etiology of Atrophic Rhinitis was published in the Fraenkel Festschrift Number of The Annals, December, 1906.]

Microscopic examination of the secretion in cases of atrophic rhinitis was first performed in the year 1882 by E. Fraenkel, by means of Gottstein's tampons left in the nasal cavities from two to six hours. He found cells and microorganisms after two hours, more numerously after six hours.

According to Dobell this secretion is made up mostly of leucocytes, partly, however, of the so-called "mastzellen." It is generally found at certain constant points on the mucous membrane, the spots spreading out and over the adjacent tissue.

Nanek draws attention to the deficiency of rhodan in the secretion of ozena. (Normally it exists in the serous glands.) Muck confirms this.

SYMPTOMS.

FETOR.—The most important symptom, characteristic of ozena, is fetor, which constantly occurs in this disorder, and distinguishes it from simple chronic rhinitis. It is so characteristic that when once met with, it will never be forgotten. It is almost insupportable in removing the crusts. I remember, among others, the case of an 18-year-old girl, whom I was obliged to receive at special hours. Notwithstanding opening of the windows and disinfection of the rooms, the odor persisted for hours and was even perceptible for days.

The odor, which is difficult to describe, is something like that of herring spawn, decaying cheese, the perspiring feet of certain individuals. In general, it differs from any other odor; for instance, that in suppuration of the accessory sinuses, syphilis or foreign bodies. This fetor makes a profession or study intolerable; association with other persons may cause neurasthenia or even suicide. What is worse, however, the patients often do not smell it themselves, but associates do (cacosmia objectiva).

As I have mentioned, this symptom is constant in ozena. It may be more or less pronounced, can increase or diminish under special conditions. For instance, according to Jurasz, it is stronger in winter than in summer—in general, during dry weather as well as in dry climates (frost and heat in southern countries). In the humid seasons of the year patients with ozena feel relatively better, the fetor being less pronounced.

Generally speaking, it increases during certain physiologic states of women—especially during menstruation. Jurasz ob-

served diminution of the bad odor during pregnancy There are also intermissions, so that for days or weeks there is no fetor. Jurasz calls the condition "ozaena periodica."

Diminution and Loss of Smell (Hyposmia and Anosmia).—To the symptoms disagreeable to patient belong diminution and loss of smell. This symptom, due to fatty degeneration of the olfactory epithelium, I noticed in my practice 440 times in 1142 cases of atrophic rhinitis; i. e., in over one-third, and anosmia in 180. Jurasz observed it 18 times in 170 cases.

The writer holds that even in the most advanced forms of ozena, smell may be intact, which is difficult to explain, but I have observed it. Once, even, I noticed hyperosmia in a very nervous, hysterical woman, suffering from typical ozena. I have also observed parosmia (cacosmia subjectiva) 10 times in my practice. Relatively, often simultaneously with diminution or lack of smell, there was diminution or absence of taste (agensia et paragensia; that is to say, in 20 cases of atrophic rhinitis).

Obstruction of the Nose.—Obstruction of the nose with difficult breathing is caused by the presence of crusts on both sides, or unilateral in cases of deflection of the nasal septum, and then always on the concave side. It occurs more often with flat nose (Jurasz). Finally it may occur in cases of accompanying affection of the larynx (laryngitis sicca).

Dryness of the Nose and Throat.—Dryness of the nose is a very disagreeable symptom in cases of atrophic rhinitis simplex as well as foetida, seu ozaena. It is dependent on the character of the secretion which tends to dry, so that patients, rather seldom employ handkerchiefs, but simply extract the crusts with their fingers, sometimes removing complete casts of the nasal cavities, which often produces bleeding.

In general, as Jurasz rightly remarks, these patients are characterized by a lack of predisposition to acute catarrhs, at which times they feel much better.

As infection of the nasopharynx and pharynx, and sometimes larynx and bronchi, exists simultaneously with atrophic rhinitis, there is added to the disagreeable dryness of the nose a sensation of dryness in the throat, which is especially annoying at night. In the morning actual vomiting may be caused by the effort to extract dried secretion from the nasopharyngeal cavity. There are also hoarseness and cough as well as dyspnea.

EPISTAXIS.—Relatively often (64 cases) I noted in my practice bleeding from the nose (epistaxis). Jurasz also mentions this symptom (6 in 170).

AURICULAR AND OCULAR DISTURBANCES.—In cases of complicating disease of the eyes and ears, as well as the accessory sinuses of the nose, there are rush (strepitus), diminished hearing, tearing (lachrymation), weakness of vision, suppuration and supra- and infraorbital neuralgia.

HEADACHE.—Headache belongs to the frequent symptoms of atrophic rhinitis, not only in the event of complications, but also in a reflex way, in the form of pressure in the region of the forehead and temples, often actual migraine. In 1142 cases, I noticed this symptom in 220, almost 20 per cent. Jurasz met with it in 23 of 170, 13.5 per cent.

VERTIGO—Vertigo I observed 10 times, of which 6 probably depended upon auricular processes.

APROSEXIA.—Aprosexia, a common symptom of adenoid vegetations (Guye), I observed in 70 cases of atrophic rhinitis, of which, however, 60 cases were complicated with adenoids.

ASTHMA.—Relatively often, i. e., 28 cases, I have found asthma with atrophic rhinitis; in 18 of these, however, there were hypertrophied turbinates. In the other 10 cases the asthma must be explained as a reflex due to irritation by crusts.

OTHER SYMPTOMS.—Nasal speech—20 cases, of which 15 had adenoids.

Enuresis nocturna—4 cases, all complicated with adenoids.

Paresthesia pharyngis—20.

Neuralgia linguae—4.

Neuralgia trigemini—4 cases, one with a complicating chronic otorrhea.

Neurasthenia, especially in the form of cancro- syphilo- or phthisiophobia, relatively frequent, i. e., 48 cases. Probably a result rather than cause of atrophic rhinitis.

COMPLICATIONS.

The complications of atrophic rhinitis are, in general, frequent. Peck, author of a special contribution (Paris, 1889), found them in 21 per cent. According to this author they occur principally in the following organs: (1) Lachrymal passages and organs of sight. (2) Accessory cavities of the nose. (3) Ears and mastoid processes. (4) Larynx, trachea and bronchi. (5) Organs of digestion. (6) Nervous system.

ADENOIDS.—One of the most frequent complications of atrophic rhinitis is the condition known as adenoid vegetations. These I found in 64 of 1142 cases, more than 5 per cent.

According to Grünwald and Conetenu, adenoid vegetations predispose to ozena, so that they are of the opinion that there exists a causal relation between these two disorders. Grünwald saw recovery from ozena after operation for adenoids. Kayser, on the contrary, is of the opinion that ozena and adenoids mutually exclude each other, with which, however, Cholewa does not agree. Heymann, supplanting the view of Grünwald, goes still further; he maintains that their existence acts as a repressing influence upon the development of ozena, and advises artificially increasing their volume as a therapeutic measure, by brushing the nasopharyngeal cavity with powdered capsicum.

HYPERTROPHIED TURBINATES.—I have observed hypertrophy of the nasal turbinates quite often, 60 times in 1142 cases, over 5 per cent. The middle turbinates alone were affected in 28, the inferior turbinates alone in 22 and both in 10, usually the middle being affected on one side and the inferior on the other. Jurasz also noted these hypertrophies 8 times in 170, 4 being of the middle.

These statistics confirm the opinion of most authors that atrophic rhinitis is a result of the hypertrophic form (John Mackenzie, Gottstein, Schaeffer, Moure). Besides the clinical facts in favor of this view, there are histologic researches showing that under the microscope both the processes have been observed together (Flesh, Sieur, Jacob).

Adherents of the theory of the heredity of atrophic rhinitis (ozaena neonatorum), Schirmansky, for instance, regard this latter as proof to the contrary. John Mackenzie, however, rightly calls attention to the fact that even in such cases we cannot be sure there was no catarrh in utero.

Demme saw the transition of the hypertrophic into the atrophic form, which I also had occasion to see in some cases. Fisher, Williams and Gruber also found both forms simultaneously.

There are opponents of this opinion, such as Wyss, Heymann, Roth and Mulhall. The latter advances the following: (1) In most cases of hypertrophy there is no subsequent atrophy. (2) There are no cases in which, in one and

the same person, both forms have been established. (3) Atrophy always occurs in children and hypertrophy in adults. (4) Atrophy occurs oftener in females. Mohrmann, again, maintains (incorrectly, however) that we have no histologic proofs. Valentin and Kayser are of opinion that the disease is congenital (hereditary). Rethi thinks that both opinions are correct—that atrophic rhinitis may result from the hypertrophic or be independent.

HYPERTROPHY OF TONSILS, DEVIATION OF THE SEPTUM, ETC. I observed hypertrophy of the palatine tonsils in 60 cases, and hypertrophy of the lingual tonsils in 42, in 20 of which there existed simultaneously hypertrophy of all of Waldeyer's ring.

Deviation of the septum, ridges and spurs occurred in 48 of my cases, which may have some significance in the etiology of atrophic rhinitis, the concave, i. e., the wider, side being nearly always the one affected by the atrophic process.

Further, I noticed 16 cases with caseous tonsillitis; 10 cases with nasal polypi, 32 with disfiguration of the nose (nez de mouton, saddle-nose), 10 of which were syphilitic; 20 with perforation of the septum. Jurasz observed the latter in 10 of 170 cases. I had 4 cases with ulcus rotundum v. perforans, probably due to scraping off dried secretion with the fingers on the antero-inferior aspect of the septum; i. e., on the so-called "locus Kiesselbachii," or "xerosis" of Zuckerkandl—sites of nasal hemorrhage. Bulla ossea of the middle turbinate I observed in 2 cases, and acne rosacea in 2. Eczema nasi occurred frequently, 56 in 1142 cases; Jurasz noted it in 5 of 170.

OTHER PATHOLOGIC PROCESSES.—Of other pathologic processes I have noticed: Corpus alienum nasi 2 cases. fibroma or angioma uvulae 2 cases; lingua nigra 1 case (Wyss 2 cases); tonsillitis leptothricia 2 cases; pharyngitis granulosa et lateralis s. retroarcuatis 14.

As regards catarrh of the pharynx and nasopharynx (rhinitis posterior et pharyngitis chronica), it occurred in nearly all my cases and was of the same character; i. e., an atrophic dry catarrh. Jurasz noted the same (55.3 per cent) as well as Siebenmann, Loewenburg and Guye (constant).

Disturbances of digestion (gastritis dyspepsia) were seen by Peck. He also noted anomalies of the nervous system. Desimani saw 2 cases of epilepsy cured after recovery from ozena.

LARYNGITIS ET TRACHEITIS SICCA.—According to Semon, affection of the larynx and trachea with the atrophic process (laryngitis et tracheitis sicca), at least in England, belongs to the rare complications. For the most part it is secondary; according to Fisher, it is always so. I have observed laryngitis sicca as a secondary complication in 44 of 1142 cases, in which tracheitis occurred in 4.

Some authors (Luc, Hunter, Mackenzie, Massei, Moure, Hope, Mendel, Baginsky, Zarniko, Hitt, Wagner, Chauveau, Hamilton, Grönbeck and G. de la Notte) observed primary laryngitis as well as tracheitis sicca, seu ozaena laryngotrachealis. Luc in such cases, three in number, found the diplococcus of Loewenberg in the secretion of the larynx and trachea. The characteristic symptom of such cases is fetor ex ore, the nasal cavities being quite healthy. I also had occasion to observe 4 such cases of primary laryngitis s. tracheitis sicca (ozaena laryngotrachealis). In 39 cases I noted simple chronic laryngitis and 4 acute.

Jurasz also met with this complication relatively often. Of 170 cases he found laryngitis sicca in 8, simple laryngitis in 8 and tracheitis sicca in 1.

In 87 cases there were bronchial symptoms (chronic inflammation). Paresis laryngis I noted in 24 cases. Jurasz reports 3 such. Hoarseness without changes in the larynx was observed by Seiss, Langrange, Gougenheim (overextension of the cords) and Moure (disturbance of innervation of the expiratory muscles).

Chauveau saw hemoptysis, simulating that of a tubercular process in the lungs, in a case of ozena, which I also had occasion to observe in two cases.

Stenosis of the larynx (blenorrhea Stoerki) was seen in this disease by Suketowski and Herynz.

AURAL COMPLICATIONS.—I have seen ear complications exceedingly often, 166 of 1142 cases; i. e., more than 14.5 per cent. Zaufal was first to draw attention to this fact. Wyss in 60 cases noticed this complication 47 times. Likewise, Williams, Noquet, Berthold and Peck. Only one, Loewenberg, is of contrary opinion; i. e., that aural disturbance is rare in this disorder.

In the pus of cases of chronic otorrhea accompanying atrophic rhinitis, Maclay and Viollet found the bacillus mucosus of Loewenberg-Abel. Ferrari, who occupied himself espe-

cially with this question, found them in 44 of 430 cases.

Of my 166 cases of ear disease the greater part, 74, were chronic purulent otitis; the remaining cases include simple middle ear catarrh (22), of the Eustachian tubes (20), acute suppuration of the middle ear (6), acute catarrh of the middle ear (4), cholesteatoma (3), caries of the mastoid process (6), lues (5), tuberculosis (4). Quite often I met with cerumen, finding it in 20 cases. There were 2 cases of deafmutism.

Jurasz noted aural complications in 12 of 170 cases of ozena.

Ocular Disturbances.—These are also relatively frequent in atrophic rhinitis. I noticed them in 80 cases, over 7.5 per cent. Jurasz had 4 in 170.

In my cases the nasolachrymal canal was particularly affected, simply by continuity, resulting in dacryocystitis with tearing 30 cases, and blenorrhea of the lachrymal sac 20 cases. There were 10 cases of blepharoconjunctivitis, 8 of conjunctivitis and 2 of scrofulous keratoconjunctivitis. Of the rarer pathologic processes I noticed empyema palpebrarum 1, cataract 2, nystagmus 1 and muscae volantes 6.

Williams often observed affection of the lachrymal ducts in this disorder. Gruber in 30 cases of dacryocystitis noticed ozena in 19 and atrophic rhinitis in 8. Ulcer of the cornea was seen by Trousseau and by Bick, optic neuritis by Sulzer. Peck saw dacryocystitis, conjunctivitis, ulcer of the cornea and optic neuritis.

Accessory Nasal Cavities.—The accessory cavities of the nose are often affected, mostly as empyemata. In my cases I observed this complication 86 times, about 8 per cent. All of these cases were proved by transillumination as well as by other methods of examination.

Of these cases 20 were suppuration of the antrum of Highmore, 12 both, 14 the right and 16 the left. The frontal sinus was affected in 9 (in 4 cases both, in 3 the left). In 6 cases there was empyema of the frontal and maxillary sinuses. The ethmoid cells were affected in 20 cases (caries and empyema) in combination with empyema of Highmore's antrum 4. There were 4 cases involving the sphenoidal sinus, and in 1 case the maxillary, frontal, sphenoidal and ethmoidal were all involved.

What is the connection between ozena and empyema of

the accessory sinuses? Is the theory of Grünwald, that ozena is of sinus origin, correct?

This question has already been solved negatively by the writer. Although both these diseases often occur simultaneously, as shown by my statistics above, the causal connection, in Grünwald's meaning, has not been proved. For instance, in my observation of 290 cases of atrophic rhinitis in which transillumination was used, I could not find any changes in the nasal accessory cavities. I have also remarked that the majority of authors think Grünwald's theory is incorrect. According to Luc, empyema of the maxillary antrum often simulates ozena.

CLINICAL PICTURE OF ATROPHIC RHINITIS.

Above all ex aspectu, the general appearance of the face is striking in this disease. It is pale, swollen. The nose and lips, especially the upper, are also swollen. In a word, the symptoms are those of scrofulous patients.

Usually these patients exhibit a bad general state (Loewenberg), though not always. Very often we meet with quite healthy looking persons.

The external form of the nose frequently shows more or less pronounced change (Krieg). The nose is more or less diminished in volume and tilted. The nostrils are vertical, or, what is more frequent, the nose is thick, flat at the base, more or less concave, often the typical sheep nose (32 cases in my practice). Retracted alae nasi have been observed (Stewart).

Generally the changes of external appearance of the nose are frequent in atrophic rhinitis, especially the fetid form, ozena (Patiquet, Valentin). Some authors are of contrary opinion (Noquet, Cozzolino). Even, Voltolini, for instance, maintains that he has never seen any change in the external configuration of the nose.

ANTERIOR RHINOSCOPY.—Upon anterior rhinoscopic examination we see the nasal cavities filled with more or less dry secretion (crusts) seemingly covered with meal-dust, dirty, green, sometimes blackish, tenaciously adhering to the anterior end of the middle turbinate, or, more rarely, the inferior turbinate, the nasal septum and posterior part of the nasal cavity.

Sometimes these crusts hang inside the nose like stalactites. There is also more or less fluid secretion, grayish mucus or yellow purulent.

After removing these crusts by careful (!) irrigation of the nose, or by using the forceps, the nasal cavities appear wide, one on the concave side of the septum, generally wider than the other.

On minute examination it is evident that this increase of volume of the nasal cavities is caused by a more or less pronounced atrophy of the turbinates, especially the inferior and partly the middle. The turbinates appear as mere ledges or prominences.

Sometimes the middle turbinates adhere to the nasal septum, in case it is deflected, or when their anterior ends are hypertrophied, which, as I mentioned, happens pretty often.

This condition of the cavities permits seeing by anterior rhinoscopy details which are normally impossible to see, as, for instance, the entire hiatus semilunaris with the openings of Highmore's antrum and the frontal sinuses, as well as the posterior wall of the nasopharynx with its so-called Passavant's prominence, the lateral walls with the openings of the Eustachian tubes, the ostia of the sphenoid sinus, the vault of the pharynx with more or less of Luschka's tonsil and the movements of the velum palati in speaking and swallowing.

Atrophy of the turbinates may be unilateral (Jurasz 3 cases, myself 20 cases—12 on the left and 8 on the right side, usually in the wider cavity). Such cases of unilateral atrophy were observed also by Strübing, Semon, Siebenmann, Smit (also cases with deflected septa) and Schmiegelow. Jurasz made special observations as to the state of the turbinates in 180 cases of atrophic rhinitis. He noted atrophy of both inferior turbinates 5 times at the anterior ends and 4 times at the posterior; in 2 cases both the inferior and middle turbinates were atrophied.

In 1142 cases of atrophic rhinitis simple and fetid, I noticed atrophy of both turbinated bodies 380 times, about one-third of all. The atrophic process was especially marked at the anterior ends of the turbinates. On the middle turbinates I noticed it only 24 times, and 15 times in both inferior and middle.

As above mentioned, I observed hypertrophy of the middle turbinates rather often—60 cases. In the remaining cases the turbinates were practically normal, at least without pronounced changes.

In 35 cases there were combinations of the hypertrophic

and atrophic processes—in one-half the nose atrophy of the turbinated bodies (20 cases) or middle turbinates (15); in the other, hypertrophy mainly of the middle, and more rarely of the inferior, turbinates.

The mucous membrane may also be more or less atrophied, and here, again, there may be hypertrophic spots as well as atrophic ones. Accordingly, the mucous membrane appears glistening, pale, parchment-like; elsewhere, however, red, soft and bleeding slightly when touched.

COURSE.

The course of atrophic rhinitis, simple or fetid, is generally very slow—it is chronic par excellence. The disease lasts many years (40—Semon). As I have said, there are ameliorations and deteriorations, even intervals of a relatively healthy condition (Jurasz).

I have observed one case, that of a young unmarried woman, in which the very obstinate ozena disappeared after marriage, although the contrary may occur.

DIAGNOSIS.

The diagnosis of atrophic rhinitis is in general not difficult, in its fetid form, ozena. In the latter one symptom is sufficient to establish the diagnosis of ozena, namely, the fetor which is so characteristic that it is hard to make a mistake.

Although there is fetor also in other purulent pathologic processes in the nose, for instance, empyema of the accessory cavities, foreign bodies, syphilis, etc., nevertheless, as I have mentioned, this fetor is quite different from that of ozena.

In view of the parasitic origin of atrophic rhinitis, a bacteriologic examination showing Loewenberg-Abel's bacillus mucosus may also confirm the diagnosis; likewise a microscopic histologic examination showing cornification of epithelium, the so-called metaplasia (Siebenmann). This latter method (excision of the middle turbinates and examination under the microscope, is advised by Sporleder as well as by Grosskopf.

Chemical examination of the secretion in ozena may assist by revealing a deficiency of rhodan (Muck).

Sometimes it is rather difficult to differentiate atrophic rhinitis from similar pathologic processes, such as so-called

rhinitis sicca anterior (Ribon and Siebenmann), xanthosis (Zuckerkandl) and the blenorrhea of Stoerk.

It is easier to distinguish this disease from rhinoscleroma, especially as the bacillus of Frisch appears to be identical with Loewenberg-Abel's bacillus mucosus.

PROGNOSIS.

The prognosis of atrophic rhinitis, either simple, or, particularly, the fetid form, is favorable only quoad vitam. it is bad, however, quo ad valetudinem completam, recovery from this disease being exceedingly rare.

Most authors are of opinion that atrophic rhinitis, especially ozena, is incurable (Mulhall, Lefferts, Gelle and Blendian, Wagner, Flatow, Krakauer, Watermann, Haviland Hall, Cholewa and Cordes). On the other hand, many writers are not so skeptical as to recovery. Of these, should be mentioned first of all the French school with Moure at its head (Noquet, Garrigue, Dessalines, Baratoux, Chazazac, Lacoarret); the Belgians (Cheval, Delie, Capart, Rousseau). In America, as well as in Germany, this view has fewer adherents (Wright, Dunn, North, Hamilton, Laker, Bram, Jurasz, Rethi and Grosskopf). In Italy, Massei, Belfanti and della Vedova—authors of "diphtheritic origin of ozena." In Russia, Schirmanski, etc.

The following authors in particular state that spontaneous recovery from ozena is possible: (Rethi, Kuttner Jurasz, Burger and Kayser), that in later life the principal symptom, fetor, diminishes and even disappears entirely (after 50—M. Mackenzie). Wright and Hugues go still further, maintaining that spontaneous recovery occurs at every age.

According to Dunn, even complete regeneration of the atrophied mucous membrane is possible—a very audacious opinion, to say the least.

All of these authors agree, however, that recovery is only possible with an abundance of patience on the part of both physician and patient during the treatment. The latter is necessarily long, lasting not weeks but months and even years (Moure, Jurasz, Wright, Grosskopf). In my opinion recovery from atrophic rhinitis is possible, but, at most, it can only be relative, i. e., disappearance of the

chief symptoms, fetor and secretion, sometimes the regaining of smell.

However, as to regeneration of atrophic mucous membrane and nasal bones, I do not believe in it.

TREATMENT.

GENERAL.—Treatment must be two-fold—local and general. This latter, in my opinion, is very important, and in every case where the general state of health is defective must be applied.

The relatively favorable results from my treatment of this disease I must ascribe to the simultaneous use of both measures, general and local, especially as the general is causal (constitutional origin of atrophic rhinitis). Kyle, Schestakow, Grosskopf and Mazazuto are, among others, of the same opinion.

It is very important to apply from childhood prophylactic remedies—general treatment. There is no doubt that in this manner we could avoid the appearance of this pathologic process at a later age.

So, for instance, beginning at the earliest age there must be the best and most reasonable hygiene, including suitable nutrition, gymnastics, sports, fresh air, avoidance of excessive mental work, etc.

As very suitable for children I regard the use of oleum jecoris aselli as well as preparations of iodin (iodid of iron by itself, or, still better, with equal parts of oleum jecoris aselli, pilulae Blanecart, etc.). Iodic salt baths (Hall, Creuznach, Ciechainek, Loomis) as well as sulphur baths (Treveren, Busk, Schinznach, Baden) may be used, especially in scrofulous cases.

At the same time the local manifestations of this constitutional predisposition, such as adenoids, hypertrophied tonsils, must also be treated—naturally in a surgical way.

If the atrophic process already exists the general treatment is also indicated, particularly iodin salts and sulphur baths.

Sea baths are warmly recommended by Friedrich. This writer holds that the humid sea climate has a favorable influence upon the dryness as well as crust formation in the nose.

Thomas R. French advises simply fresh air and cold baths, which are especially indicated for neurasthenic patients.

In cases of anemia or chlorosis in young girls good effect

is had from the iron baths of Francenbad, Krynica, Elater, St. Moritz and in cases of obesity, Marienbad. For nervous patients mountain air (Switzerland, Zakopan) must be advised.

Of drugs, besides the before-mentioned iodin and iron, there are also used with more or less success arsenic, iodin and phosphorus (Kyle) and Heyden's creosotal (Mazazotto)—24 to 30 drops in syrup or oleum jecoris aselli, t. i. d.

LOCAL TREATMENT.—I pass now to the drugs and methods locally applied in atrophic rhinitis, the number of which is legion. The methods employed in this disorder ranging from the oldest, irrigation of the nasal cavities, to the newest, subcutaneous injection of serum, all have their adherents and opponents.

IRRIGATION.—Irrigation by means of Weber's douche or the double English syringe is the oldest and until now most common therapeutic method of treating ozena. It is dangerous, however, when not correctly used on account of the possibility of infecting the ears, especially because to cleanse the nasal cavities of crusts it is necessary to employ, (1) considerable force and (2) great quantities of fluid (20 to 50 litres!—Range). In order to avoid these bad consequences (Michel, Grosskopf, etc.) we must remember three things: (1) The syringe must be held horizontally, i. e., in the direction of the inferior meatus, (2) always to irrigate on the narrower side (convexity of the septum) and (3) not to employ too strong a current.

Greville McDonald and also Hamilton advise a lying position with the head dependent.

At Schwartze's clinic in Halle the irrigation is done from behind through the nasopharynx. This is also advised by Moure, of Bordeaux, and Jurasz, of Heidelberg, with a special curved syringe having several openings at the tip.

On the other hand, it is exceedingly important to keep the nasal cavities clean, this being the basic principle in treating atrophic rhinitis (Klemperer).

NASAL BATHS.—This aim can be reached by other methods, as, for instance, the so-called nasal baths (M. Schmidt) which consist in pouring fluids into the nose by means of a teaspoon or Fraenkel's special apparatus, in order to avoid the entrance of the liquor into the pharynx or larynx.

The temperature of the fluid, which must be tepid, is of importance. Kiesselbach advises commencing with a temperature of 10 degrees R. and going up to 14 degrees R.

SPRAYS.—The application of drugs in the form of sprays by means of Hartmann's or Schnitzler's sprays, either in front or behind, is generally insufficient for thorough cleansing, although the plan has its advocates (Grosskopf, Musehold, Meyjes).

TAMPONS.—Still better are tampons, saturated with various remedies, introduced into the nasal cavities and left there for a time.

This method was employed for the first time in 1878 by Gottstein, and has been much used since then. Among others, Krieg is an ardent supporter of this plan, which he regards as the best. So also are Aysaguer, Krakauer, Seiler, Baratoux, Hamilton and Hagedorn.

The method consists in the introduction of tampons, 3-5 cm. long, by means of special sounds (Gottstein, Grosskopf) or without using the latter (Krieg, Seifert).

The tampon remains in the nose 24 hours (Gottstein) or 4 hours (Krieg) or only a half or quarter hour (Zarniko).

A modification of Gottstein's tamponnade is Bruck's, the so-called permanent one. The tampon can be wet with different remedies.

BRUSHING.—Brushing with various solutions is sometimes employed, without being efficacious, however.

MASSAGE.—Still better is vibratory massage of the nasal mucous membrane. This method was first introduced into the therapy of diseases in the upper respiratory tract in 1890 by Braun, of Trieste, and later by Laker, of Graz. Since then it has found many supporters—Garnault, Felici, Sendziak, Demme, Jankau, Dionisio, Kirchner, Krakauer, Abeeille, Pleuric, Lucae, Brenner, Sterch, Watermann, Moure, Gradenigo, Daae, Blondian, Berthold, Seiss, Burger, Boris, Brindel, Spiess and M. Schmidt. Most of them employ for massage special sounds with a wad rolled on the tip (Braun, Laker). Others advise apparatus—Daae, Löhnberg, Breitung, foot-motor; M. Schmidt, electric motor; Dionisio and Jankau compressed air.

The most conspicuous opponents of the method are Chiari and Kuttner.

As for myself, like Braun, Laker, Garnault, I, too, regard vibratory massage as the best treatment of atrophic rhinitis, effleurage and vibrations being able to stimulate the atrophied mucous membrane to renewed life. In the main with the

above method I had the best results, even relative recovery frequently, in this obstinate disease.

INSUFFLATIONS.—As to insufflation of different powders, I regard this method, like Schech and Grosskopf, as not only useless, but even harmful, since it increases the disagreeable dryness of the nose.

INHALATIONS.—Although insufflation has its adherents (Bresgen, Rosenthal, Killian, Stetter, Hemm), a more rational method, especially where there is a complicating affection of the larynx, is the inhalation by means of Siegel's apparatus. This method is recommended by Kafemann, Kuttner and Dagail.

THE DIFFERENT THERAPEUTIC AGENTS.—By the above methods have been used thousands of remedies, of which the greater part have no value, and can be discarded without loss. Of these remedies I shall enumerate the most important.

RESOLVENTS.

1. Sterilized water (Barth).
2. Hot air (Kuttner, Dagail).
3. Sodium chlorid, a tablespoonful in a litre of water (Schwartze, Grosskopf, Mulhall, Valentin).
4. Mineral waters. Iodin salts, such as Reichenholl (Grosskopf), Wiesbaden, Ems (Ciechaeinek); sulphur (Cantoret-Lagrange); thermal (Compaired).
5. Potassium chlorate (Brügelmann, Moure, Baette, Nooves—the last using it as a spray 1-80).
6. Sodium bicarbonate (Mulhall, Moure, Kuttner). (Kuttner uses it by inhalation.)

ANTISEPTICS.

7. Boric acid; 10 to 30.0 in a litre of water, or alone (Mader).
8. Sodium biborate 20.0 in glycerin 70.0 and water 30.0 as a spray (*N. Y. Medical Record*); together with carbolic acid, salicyclic acid and potassium hyp. (Lefferts).
9. Sublimate; 1 to 10000 (Schondorff, Loewenberg, Fackeldey); together with boric acid (Loewenberg).
10. Potassium permanganate; teaspoonful of 10 per cent solution in 1 litre of water (Kirchner, Schwartze).
11. Lysol (Stetter).
12. Thymol; 0.03 to 0.09 in alcohol and glycerin aa 2.0 and aqua dest. ad 30.0 as a spray; 0.3 to 0.6 in alcohol and glycerin aa 15.0 for mopping (Wright, Ledermann). 0.1 gly-

cerin and spirits aa 6.0, aqua dest. 30 (Siefert), or 20 per cent glycothymol (Cullen). Thymol, salol, salicylic and boric acid (Sieffert).

13. Thymic acid 5.0 (0.2), acid salicylic 15.0 (0.5), acid boric 5.0 (3.0), salol 15.0 (5.0), talcum powder 25.0 (8.0), for insufflation (Ed. *Phila. Medical Journal.* Tissier).

14. Ichthyol (Ertler-Phillips). 5 per cent in creolin for brushing; 2 to 5 per cent as spray; 25 per cent for brushing (Knight); in petroleum 50 per cent, ammonium sulpho-ichthyolate, 40 to 60 per cent (Strübing).

15. Creolin (Plesskopf), tampons; creolin-vasogen (Strübing).

16. Naphtol (Rualt) 12.0, alcohol (90 per cent) 84.0, also camphorated naphtol with vaselin.

17. Menthol—Bommier.

18. Europhen—Loewenstein, Ledermann.

19. Aristol (Loewenstein), dry; with collodion (Fasano, Sieffert, Braislin, Heymann).

20. Iodol—dry and on gauze, Flatau, Tissier; as snuff, KI with tannic acid and sodium biborate, equal parts, Turban.

21. Soziodol—Sieffert, Bresgen.

22. Iodin preparations—Baratoux; NaI by insufflation, Wolff; iodin and potassium iodid, Schroetter.

23. Cresol iodid—Peterson, Seifert; metacresolanytol, 1 per cent, Strübing; tricresoliodin, Ewing.

24. Resorcin—Tissier, John North; with cocain, 10 per cent, Leland.

25. Aluminum aceto-tartrate—10 per cent, Bartholow; dry, Schaeffer, also in 50 per cent solution; a teaspoonful in a half to one liter; or 20 per cent, 10 drops in a glass, Krakauer. This remedy also gave me satisfaction.

26. Balsam of Peru—in equal parts with glycerin, Cohn, Seifert, Ebstein, Rosenbach. Upon tampons I often found it efficacious.

27. Glycerin—Roe; with vaselin, Seefers.

28. Oleum terebinthinae—on tampons, Malacrido.

29. Eucalyptus—Brown, 20 or 30 drops in an ounce of vaselin or lanolin.

30. Formaldehyde—Geo. L. Richards, 5 to 10 drops of 40 per cent in a glass; Knight.

31. Petroleum—petroli depurati 40.0, strychnin nitrate 0.02, ol. eucalyptus odor citrici 0.50, Bobone.

32. Hydrogen peroxid—Hope, Flatau, Watermann, Kyle, Seiss, Rosenthal.

33. Methylene blue—1 to 10 sodium sozoiodolate, Bresgen; 25 to 1000 Bennett; Moir.

34. Phenol sulphoricinate—30 per cent, Grosskopf; phenol sodium sulphoricinate, Dreyfuss.

35. Fl. ext. hydrastis—Bartholow.

36. Saponin—12 per cent, Valentin.

37. Zinc stearate—25 per cent, Gibb.

38. Listerine, an American antiseptic—1 to 10, Hall.

39. Acetanilid—Hubbard.

40. Creosote—5, alcohol 10, glycerin 40, Ferreri; 1 to 4 of glycerin, Desimeoni.

41. Citric acid—Flamur, Somers (75 per cent); Zaalberg, as powder with sacch. lactis.

42. Orthochlorphenol—10 per cent, Nichols.

43. Antozone—1 per cent, Brown.

44. Ozone—Rice.

45. Mustard oil—Kyle, 1 to 500.

46. Atrophin sulphate—Noquet, Baratoux.

47. Silver nitrate—Seiler, in powder; Estien, Mayes, 2 to 25 per cent in spray.

48. Zinc chlorid—1 to 10 per cent, Estein; zinc stearate, Leyler, Bresgen.

49. Trichloracetic acid—Bronner, Stein, ½ to 10 per cent in ozena, ½ to 1 per cent in atrophic rhinitis; 20 per cent, North, Rethi.

50. Chromic acid—Rethi.

51. Carbolic acid—Jurasz on tampons.

52. Calomel—with starch, 12 to 15.

53. Nosophen—M. Schmidt.

Sometimes several agents are included in one formula, as:

Acid carbolic		1.3
Aristol		2.6
Ol. ricini		
Ol. cubeb	aa	4.0
Camphor		
Menthol	aa	2.0
Thymol		
Ol. eucalyp	aa	6.0
Albolene	q. s. ad	128.0

M. Sig. spray. Wenzel.

Of the latest remedies and methods I must enumerate:

1. Intramuscular injections of iodin (1-3 aq.), Gradeingo.
2. Interstitial injections of vaselin (60 per cent) into the inferior turbinate, Brindel.

Injection of paraffin, Brindel, Compaired, Lake, Baratoux, Brockaert, Siess. This method is similar to that employed by Gersuny and others for "saddle nose," where it must be used with care. Brindel noted two cases of phlebitis. Flatau and Delic are opposed to this method.

SERUM TREATMENT.—In 1900, the Italian authors Belfanti and della Vedova, after bacteriologic investigations which showed the presence of pseudo-diphtheria bacilli in cases of ozena, suggested the use of antidiphtheritic serum. In 32 cases they had 16 recoveries, 7 were greatly improved, 4 were respited and 5 slightly improved. The opponents of this method are Ambrosini, Lombard, Avalan, Catherina, Samurta, Habermann, Kayser. Mediocre results were obtained by Gradenigo, Massei, Masini, Longhini, Grazzi, Farci, Ostino, Borio. Garnault in one case after injection observed acute articular rheumatism. Torres used normal horse serum with small success.

Generally speaking, the method found adherents only in Italy and while it was novel; very few in other countries (Compaired, Mouret, Cathelin, Lautmann, Molinie, Cozzolino, Nowtalo, Ablow and, to some extent, Frankenberger).

ELECTRICITY.—Electricity is rarely applied in the treatment of atrophic rhinitis and ozena. Faradization has its advocates in Garrison, Ralph W. Seiss and Delavan. Hartmann used galvanic electricity.

GALVANOCAUTERY.—The galvanocautery is rarely used in this disease (Noquet, Abeille, Retzi). Garrigeu, Desarienes, Mercié and Aysaguer regard the method as too painful.

ELECTROLYSIS.—Electrolysis has often found application, having advocates and opponents. The method was first used by Desarienes in 1884, afterward by Bryson Delavan and Kafemann. It has its warmest adherents in Belgium in Chevral, Bager, Joulsin, Capart and Rousseaux. Chevral, for instance, claims that he obtained by this method, which he calls "interstitial cupric electrolysis," 91 per cent of recoveries. The needles are introduced into the middle and inferior turbinates, the positive pole being copper, the negative steel. The action is bactericidal. According to Cobb, the materials of which the poles are made have no significance.

Other supporters of the method are de Roaldes, Hugues, Thomas (who advises a weak current, more than 15 milliamperes often being painful), Rethi, M. Schmidt, Moll, Dagail, Braat, Bride, Scholl, Bouronvillee, Marsip, Yonge and Massini.

As opponents, however, of this method, who are numerous, I must number the following: Goris and Delsaux, Schiffers, Hudelsohn, Schech, Haindl, Eckstein, Hajek, Chiari, Hennebert, Anehe and Brindel, Magnon, Mouré, Zaalberg, Burger, Frederiks and Grosskopf.

I, also, am not a partisan of electrolysis in the treatment of this disease.

SURGICAL TREATMENT.—The surgical treatment of atrophic rhinitis and ozena finds few supporters. Cholewa advises fracturing the bones of turbinates. Cordes incises the turbinates. Tackeldey and Berliner extirpate the anterior hypertrophied end of the middle turbinate. Mackenzie advises curettement of the nasal cavities, and Guye of the nasopharynx.

MECHANICAL TREATMENT.—Saenger narrowed the nasal orifices with his nasal obturator. Kafeman's obturator also serves as an inhaler. Barth, for this purpose, used the hygroscopic wad, and Magdelung two pigeon feathers. Flatau employed pieces of ivory, and Claverdat, as well as Fodenat, caoutchouc tubes.

I must mention a curious fact: the favorable influence of erysipelas upon ozena—2 cases of Francesco, 1 of Somers and 1 of Levi.

Here, too, belongs a strange case reported by Spencer Watson: recovery from ozena after gonorrheal infection.

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LV.

A REPORT OF A CASE OF FIBROMYXOMA, INVOLVING THE LEFT SUPERIOR MAXILLA, INCLUDING THE ALVEOLUS, FLOOR, ANTERIOR AND POSTERIOR WALLS, AND OF SEVEN YEARS' DURATION.

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New York.

The knowledge of the life history of this peculiar form of growth appears to be very limited, and after considerable research I was unable to find any literature to assist me in the care of this patient. It seems to me that the duration and the persistent recurrence after repeated attempts at removal with curette and cautery, and the fact that it could not be classified as an encapsulated tumor, for it ramified in all directions and completely absorbed the bone in many places, point to a much more serious condition than is generally accepted for a fibroma. At the time of my operation there was decidedly more myxoma present than fibroma, and judging from the history given by the patient I believe that has always been the case. The freedom from pain, the absence of any tendency to suppurate (the surface always healing over after previous operations), and the lack of any lymphatic involvement contraindicate malignancy; but notwithstanding these facts, the ramifying nature, the recurrence and the gradual loss of weight of the patient seem to indicate a serious condition the true nature of which we have not yet grasped; and it is with the hope of learning more of its nature that I present this, to me, most interesting case.

The patient, Miss H. M. H., was brought to my office on February 2nd, 1907, by Dr. A. F. Bauer, a former student of mine at the dental college, with the following history: Family history excellent, both parents and two brothers being alive and in good health. Several years ago she suffered with menorrhagia and was cured by curettement. With that exception her history was good, and she had never been ill to her knowledge.

The following letter is the patient's own description of her case: "About the fall of 1900, while having some dental work done by Dr. F., of New York, I called his attention to a wisdom tooth that was troubling me. He lanced it, and later, I believe, he lanced it a second time. In February, 1901, I consulted a dental surgeon of Brooklyn in regard to having a tooth straightened. He at once noticed a small enlargement over or near the wisdom tooth, and called my attention to it, saying it ought to be attended to. I believe he then removed the tooth. Upon my return home I saw our family physician of Newark, in whose hands we placed the matter for treatment. In February, 1901, he sent some of the growth to the Board of Health of Newark, N. J. They pronounced it to be not malignant. The physician continued his treatment of it, cutting out at times considerable masses of it, hoping thus to check or destroy the growth. For a time it did not seem to be enlarging and it caused no discomfort, so little attention was paid to it. But in April, 1904, it became worse and was again treated when it was necessary to remove another tooth. From that date until February, 1907, it was not treated. March 22nd, 1907."

For two years nothing had been done, and as there was no pain the patient did not realize the serious nature of the condition until she consulted her dentist again, who immediately advised her to see a surgeon and referred her to me. For some months she had been unable to masticate on the left side, and occasionally had some dull aching on that side, but otherwise was not inconvenienced. She had at times noticed some puffiness under the left eye, but close questioning did not elicit any history of nasal discharge at any time. There had been a slowly developing mass appearing under the malar bone, but not until recently had it caused the left side of the face to appear fuller than the right. As said before, there was no actual pain, but at times there had been some dull aching on that side. From without the tumor felt hard and unyielding. Examination of the mouth revealed a great thickening of the alveolus from which the last two molar teeth had been extracted. The thickening extended forward to the canine tooth and inwardly over the palate process to within one-half an inch of the median line. Externally it bulged out forming a tumor the size of half an English walnut, with a hard bone-like wall, which extended upwards to and

apparently involved the malar process itself. Along the free border of the alveolus the mass felt soft and fluctuating and appeared somewhat translucent. The first molar and the second bicuspid were very loose in their sockets owing to the absorption of the alveolar bone tissues. An X-ray photograph shows this absorption very plainly. The nose was found to be in perfect condition and also the throat. Transillumination was negative, both antra showing light. On February 11th, 1907, under cocain and andrenalin anesthesia administered hypodermically, I made an incision along the free border of the alveolus, expecting to find a cyst. I found the fluctuation was due to the presence of a large amount of a semi-solid gelatinous mass, grayish pink in color, and I removed some of this for microscopic examination. The molar was then extracted and a probe was readily passed into the antrum through a root, it being found full of a soft growth. There was very little hemorrhage. The wound was packed with iodoform gauze, and the patient returned to her room to await the report of the pathologist. This was received on the 14th, and was as follows: Fibrous and epithelial hyperplasia in one piece of tisue submitted. In another piece, there is a matrix of homogeneous groundwork taking hemotoxylin stain, and in it are a number of finely wandering embryonic connective tissue cells. Diagnosis: Fibromyxoma.

DR. J. WRIGHT, Pathologist.

On the strength of this report I advised complete removal, and on February 26th this was done at the Manhattan Hospital, with the assistance of Drs. H. Smith and G. H. Ward. The operation was performed through the mouth, the floor, anterior wall and most of the posterior being removed in reaching the limit of the growth. It extended into the apex of the antrum, but in this region it was not attached, seeming to shell out rather easily, and the roof and inner wall of the antrum did not appear to be involved in the growth. Having dissected the periosteum from both the anterior wall and the palate process, these were removed together with the entire alveolar process up to the lateral incisor. The greater part of the palate bone was removed, exposing the muscles and opening the zygomatic and even the pterygoid-maxillary fossae, the delicate fatty tissue protruding into the wound in these regions. Having avoided the tuberosity and the posterior wall until the last, on account of the palatine arteries,

there was but little hemorrhage, the growth itself being singularly non-vascular. The involved bone was very soft, as though absorbed, and was easily removed with curettes. The growth also seemed to shell out in large masses, especially where the myxomatous tissue predominated. The greatest difficulty was found at the tuberosity and along the posterior wall, but this was overcome by using a large, powerful, round fenestrated tonsil punch, of German make, which cut through the softened osseous tissue with ease. In all, I should say, the growth weighed between two and three ounces, though possibly more. No opening was made into the nose or the orbital cavity, and fortunately the soft palate was not involved, so that there was no opening in that region. The cavity was packed with iodoform gauze, no attempt being made to introduce sutures. Dr. Wright reported again that the growth was fibromyxoma, with no evidence of sarcoma.

Subsequent history uneventful. Three months after operation the extensive cavity had entirely closed with apparently healthy tissue. The patient had gained in weight, and was being fitted with an endenture.

The literature on growths of this nature is exceedingly scant, as said before, being merely mentioned in the classification of tumors in most text books. The American Encyclopedia of Medicine says about them: "Fibromyxoma usually occurs in the region of the alveolus. When arising from the alveolus adjacent to the antrum it may project into that cavity and appear to be growing from it. These fibromata are considered by Bland Sutton to be of the same origin as odontomata. Some people consider that they may result from chronic inflammation around the apex of a tooth. Garrettson, Marshall and Grant, in their books on the surgical diseases of the jaws—books that are used as text books in the dental colleges—make no mention of these growths. Dr. Wright has written extensively on nasal growths which have been generally regarded as of this nature. Posey and Wright do not mention it. Kyle mentions it as appearing in the nose.

The most extensive description that I could find, however, is in The American Practice of Surgery, by Drs. Bryant and Buck:—"Myxomata are tumors of the mucoid character. Structurally they are composed of cells floating in a homogeneous, semi-fluid mucinous matrix. These cells are mononuclear, bipolar or stellate, and provided with more or less

elongated protoplasmic processes which interlace freely. The intercellar substance varies in amount in different tumors and in different parts of the same tumor. When abundant, it gives a characteristic gelatinous, semi-fluid, somewhat translucent appearance to the growth. Myxomata are grayish, or pinkish gray in color, owing to the presence of blood vessels that are more or less distinctly visible in the substance. On section, a jelly-like or ropy substance—mucin—exudes, which is not soluble in water, and gives a whitish precipitate with alcohol or dilute acetic acid.

Myxomata are rarely pure in type, but are usually combined with other tissues of a homologous nature. Mucoid tissue is closely related to fibrous tissue. The truth of this is evident when we remember that in the fetus the fatty tissues and fibrous tissues are first blocked out in mucoid material. Consequently, myxomata are commonly met with in the same regions from which fibromata and lipomata also spring. Further, mucoid tissue occurs in the adult body only in the vitreous humor of the eye, from which structure myxomata never develop.

Myxomata, therefore, always exhibit a certain deviation from the tissues in which they are found. In other words they are heterologous within narrow limits. The embryonic character of the cells also explains the well known instability of the tumor, which, though classed among benign growths, has a tendency to take on malignant action, the transformation occurring in the direction of the sarcoma.

True myxomata have to be distinguished on the one hand from tumors, such as fibromata, sarcomata and carcinomata, that have undergone secondary mucinous degeneration; and on the other, from growths that, owing to vascular disturbance, have become edematous. Myxomata (fibro and lipo) may originate in the connective tissue of the skin, fascia, periosteum, mucous membranes, and muscle sheaths; in the subcutaneous and subserous fat, the bone marrow, and, occasionally, in the mamma, salivary glands and testis.

Myxomata of the mucous membranes occur singly or as multiple primary tumors. The ordinary mucous polyp of the nose is a good example of these growths. Myxosarcomata, so-called, are due either to a cellular transformation of a simple fibromyxoma, or to the mucinous degeneration of a sarcoma. They behave as sarcomata and form metastases."

This last account, as will be seen, gives an excellent description from the laboratory standpoint, but does not deal with the history of the growth, its effect upon the general health of the patient, the prognosis, or the best method of dealing with it. In this case we have a tumor of at least seven years' duration, practically painless, with no glandular enlargement, but extending out in many directions, apparently non-encapsulated, and destroying the bone tissues which it invaded very much as would a malignant growth. Again, it has repeatedly healed after curettement and has never shown any tendency to ulcerate, both of which facts rather disprove the thought of malignancy. Dr. Wright's report, having been made after examination of several pieces of the growth, also indicates the benign nature; and yet the question is, where can we draw the line between benign and malignant tumors? It seems to me that any growth that can so effectually absorb the bone tissue, that returns repeatedly after operation (the thoroughness of which, however, I cannot vouch for), that appears to be without capsule and ramifies in bone, muscle, open space and fibrous tissue alike, does not belong in the benign class, and should be considered more seriously than has been done up to the present time. Bland Sutton's theory of a pericementitis being the cause or the starting point may seem to be borne out in this case, as its history of wisdom toothache preceded the growth. Dr. Cramer first noticed it while filling a cavity elsewhere and advised that it be attended to. If there is no recurrence after this extensive removal, it will be very easy to fit an obturator over the remaining cavity, with attachments to the opposite side, and with a complement of teeth that will give use for mastication, and at the same time will support the cheek if there should be much sinking in.

LVI.

A REPORT OF ELEVEN CASES OF CEREBRAL COMPLICATION DUE TO ACUTE AND CHRONIC SUPPURATION OF THE MIDDLE EAR. WITH COMPLETE ANALYSIS OF FIVE.*

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San Francisco.

In 53 mastoid operations, 17 of which were for acute mastoiditis, 36 for chronic suppurative otitis, I have found 11 cerebral complications divided as follows:

Extradural abscess, 6.
Serous meningitis, 2.
Purulent meningitis, 1.
Infectious sinus thrombosis, 2.

I wish to say at this place that all the cerebral complications developed as the result of delayed operation, and none of the cases that were operated early had complications at all. The chronic suppurative otitis cases that developed cerebral complications had cerebral manifestations prior to operation.

Extradural abscess and perisinus abscess I group in the same class. Anatomically the same, clinically they differ.

Four of the cases were perisinus abscess. Two of this group were further complicated by other cerebral affections and will be reported in full. One case of perisinus abscess with granulation tissue on the sinus was complicated by a Bezold's abscess. One case of perisinus abscess without any other complication. Two of the cases were from acute infections, one of which followed fracture of the base of the skull.

The remaining two cases were the result of chronic suppurative otitis.

*Read in the Section of Ophthalmology, Otology, Rhinology and Laryngology, California State Medical Society, April, 1907.

Two cases of extradural abscess were in chronic suppurative otitis. One case had such symptoms that an extradural abscess could be suspected; and was found at operation by a small fistulous communication. The other case was accidently discovered by uncovering the dura in the middle cerebral fossa.

Two cases of serous meningitis from chronic suppurative otitis.

One case of purulent meningitis from chronic suppurative otitis.

Two cases of infectious sinus thrombosis in acute suppurative otitis.

CASE 1. Female, aged 21. Had ordinary diseases of childhood. Acute otitis following scarlet fever at the age of 8. The discharge continued uninterrupted for two years. Adenoids were removed and drops were used in the ear which remained perfectly dry for one year. Since that time the ear has discharged more or less. Examination: Weber lateralizes to good ear. Schwabach somewhat shortened. Rinne right ear positive, slightly shortened bone conduction. Rinne left ear negative, considerably shortened bone conduction, very much shortened air conduction. Right ear whisper 25 feet; left ear whisper on contact. Acoumeter on contact. Right ear apparently normal. Left ear caries of the attic wall with a fissure extended into the same. Some granulation tissue about this fissure with the tendency to bleed on manipulation with probe.

January 19, 1905, radical operation, closing by Körner flap. Posterior wound healed by primary union. On removing the periosteum from the mastoid, the bone showed a dark blue color. This was produced by the carious necrotic mass of the mastoid cell. The outside shell of bone was more porous than under normal conditions. The hammer and incus were almost completely destroyed by caries. There was a fistula posteriorly and below the facial nerve which could not be eliminated as it would have destroyed the facial nerve. However, it was curetted as well as possible by the use of straight and curved curettes. I wish to direct particular attention to this lesion, as I consider it wholly responsible for the symptoms that will be recorded later. The wound was dressed every second or third day. The patient complained of so much dizziness and headache and pain on the side of the head that she remained in the hospital for thirty days. I attribute some of the headache to a compound astigmatism which was partially

relieved by the continuous use of her glasses. While in the recumbent position she was not dizzy. When she assumed an erect position she would become very dizzy and at one time fell from her chair. This can be accounted for in but two ways, first, that of injury to the semicircular canals at the time of operation,—or secondly, to an infection which I believe most likely took place by way of the fistula which I described before.

Twenty-four hours following operation she was reported by the nurse to be delirious. This happened two or three times in the course of that many days and she complained of headache, and soreness about this side of the head which gradually subsided. When she began to walk her gait was that of a person with a fractured pelvis. She is a highly sensitive, hysterical woman, and I attributed part of the cerebral manifestations to her mental condition. The eye background was perfectly normal.

March 27, 1905, the ear absolutely dry; hearing unimproved.

May, 1905, seropus began to discharge from this fistula and continued until September, 1905, at which time I again performed a curettement. This was treated antiseptically until November, 1905, when she was again discharged as cured. Since the recovery from her primary operation she has had no cerebral symptoms of any kind. March 2, 1906, complains of intense headache over this side of the head and dizziness a great deal of the time. The whole of the temporal bone on this side was tender to pressure and the slightest percussion would elicit excruciating pain. At this time there was a serous discharge from this fistula, which in the course of two weeks disappeared entirely. The cerebral symptoms continued with acute exacerbations, at times so severe as to require morphin. Her pulse repeatedly reached 50, full and strong. She had some vomiting which was probably due to the morphin. Examination of the eye negative. Physical examination negative.

At repeated consultations operative procedure was recommended by all but one physician, who maintained that it was due to a nervous manifestation.

May 15, 1906, patient entirely well. Cerebral symptoms entirely gone.

Diagnosis.—Serous meningitis by way of the fistula to the semicircular canal and the aquaeductus vestibuli. This will explain the cerebral symptoms that followed the primary opera-

tion as well as the cerebral symptoms that followed in the later infection. During the first infection the cerebral symptoms were scarcely sufficient to warrant further operative procedure, because they seem to improve almost daily after the second or third day. However, when we are confronted with cerebral symptoms later, and the only possible source of infection is by this fistulous communication, it must be admitted that it was by this route. Furthermore, the patient had an association of cerebral symptoms, such as dizziness, headache and localized pain on the affected side, which should always lead you to suspect cerebral complications, especially when all the mastoid cells had been removed. If it is not today, it will be in the near future considered conservative surgery to open and explore.

My diagnosis prior to her recovery was different. At this time I thought she had an extradural abscess or brain abscess, with a decided leaning towards extradural abscess of the posterior brain fossa by way of the semicircular canals and the aquaeductus vestibuli. No doubt the infections travelled this route, but was a nonbacterial invasion. I wish also to call attention to the fistula which was discovered at the primary operation and which has apparently been responsible for the infection that followed. Should it have been destroyed entirely at the sacrifice of the facial nerve, to remove all possible source of cerebral affections to follow? Or should we trust in Providence, as I did in this particular case, and almost lost my patient?

In conclusion I will say that by early operation in cerebral affections, you may cut short or eliminate entirely your source of infection, while on the other hand, if it is allowed to remain, there is but one termination, and that is death with but an occasional exception, such as I have illustrated.

CASE 2. E. H., male 28 years, clerk by occupation. Dr. Rene Bine, of San Francisco, very kindly furnished me with the medical history of this case, as well as the working out of the various nervous manifestations. Had measles at 17, which were complicated with pleurisy. For two years was in the tropics in good health. On his return to San Francisco three years ago had malaria. For the last two years he has been about San Francisco; he has lost 30 pounds in weight during the last three years. Present weight 128. Height 5 feet 7½ inches.

Present illness began about three weeks ago with cough, especially at night, and progressive weakness. Appetite good until one week ago. He had no night sweats, no chills, but a chilly feeling when out of the sunshine. No diarrhea or constipation. No digestive disturbance except vomiting following the cough. Frequent urination, part cularly during the day time. Has excessive dyspnea on the slightest exertion. No subjective cardiac trouble. Has had chronic discharge from the ear for the last two years. It seemed to start as a chronic affair. Never complained of pain or tenderness on this side of the head. About three weeks ago had to give up work because of absent mindedness. About three months ago began to see double at intervals. These intervals have increased in frequency up to the present time. The physical examination I will not record, as it was a clear picture with findings of pulmonary tuberculosis.

Examination December 29, 1905.—Pupils, dilated and unequal, respond to light and accommodation. Left dilated more than right. January 3, examination by myself. Pupils dilated, left more than right. The assistant says dilated more than three days before. Respond to light and accommodation. Apparently no vision of the right eye. Slight lateral nystagmus. When finger is held below the level of the good eye sees double, otherwise normal. No changes in the disc apparent. January 2, 1906, it was noted that the right eye could not be completely closed. The lower lid drooping and the upper lid with little resistance. Facial paralysis of the right side. The lines of the forehead and cheek were obliterated. Mouth drawn to the opposite side. Left side of face slightly flushed and warmer than the right. Neck very rigid. Regarding flexion of spine it moves as a whole. Patient can be raised to the sitting posture without flexion of the spine by the hand behind the head. Patient cries with pain when the neck is forcibly flexed. Head rotated slightly to the left. Reflexes of right arm present and normal, considerable spasticity. Reflexes of left arm normal, no spasticity. Right patella reflex slightly accentuated with spasticity. No marked Oppenheim on the right. Questionable Oppenheim on the left. Questionable Babinski on both sides. No ankle clonus. No patella clonus. Achilles tendon reflex very slight. Cremaster reflex absent on right, very active on left side. Abdominal reflex not elicited.

Examination of the Ear.—Discharge of foul-smelling pus from the right ear. The posterior superior wall of the meatus was so swollen that it formed an ellipse instead of a circle. The tympanic membrane could not be seen. Some pain on deep pressure over the tip of the mastoid.

Diagnosis.—Probable meningitis with possibility of brain abscess.

January 4, 1906, patient unconscious for the last twenty-four hours. Paralysis of the opposite side and partial paralysis of the same side. Radical mastoid operation. Pus under considerable pressure. Mastoid cells full of granulation tissue, and a cholesteatoma mass. Dura of the middle fossa uncovered very easily. Dura bulged into the operative field so that the edges of the bone could not be seen. No pulsation of the brain. Dura incised. Large quantities of serous fluid escaped. The brain still bulged into the dural wound. Was incised in three different directions, followed by the escape of considerable serous fluid, which undoubtedly came from the lateral ventricles. There was some question as to the possibility of tubercular meningitis because of the lung tuberculosis. This is of course possible. However, in a case of chronic purulent discharge from the ear with localized pain, facial paralysis and bulging of the posterior superior wall, all of which speak for pus retention, we must at once assume that such is the case and deal with it accordingly. I am not inclined to think that this was a tubercular infection of the middle ear because it began as a chronic discharge. There was no acute manifestation of an inflammation. This is in accordance with tubercular infection of the ear. On the other hand, he had measles when 17 and it may be that he had an acute otitis then and that it had been more or less quiet until two years ago. The finding of the cholesteatoma speaks for the latter. My opinion, based upon literature, leads me to believe that this latter is the correct solution. The man died twelve hours following the operation. The microscopic postmortem findings did not reveal any giant cells in the part of the dura or the brain that was examined. There was no exudate. From our findings and our lack of findings we must conclude that it was a serous meningitis dependent upon ear infection.

Case 3. L. B., male, age 12. When 5 years of age had scarlet fever, which was complicated by an acute suppurative otitis. Otherwise perfectly well. The ear discharged more

or less for the following four years. At times it would completely subside. The ear would remain perfectly dry from three weeks to two months, when he would have more or less pain in the mastoid region which would be followed by a discharge. Sometimes this pain was very slight and sometimes it was distributed to the whole of the temporal bone on this side of the head. During the four years the pain was never severe enough to keep him in bed. Many times with the subsidence of the pain the discharge from the ear would cease. During the last three years there has not been any discharge from the ear. However, he has had six distinct attacks of fever and pain in the region of the mastoid and tenderness increasing in severity with each attack. They usually last two to four days and the patient is up and about. The present attack is much worse than the former one. He has not been able to sleep for four days because of pain and tenderness on this side of the head. Temperature 102½, pulse 120. Examination: Right ear normal, left ear stands out from the head more than the right ear. Hot to touch in comparison with the other ear. The whole of the temporal bone sensitive to pressure and extremely so at the tip of the mastoid. Drum membrane entirely destroyed and part of the attic wall. The ear is perfectly dry. An unusual fullness between the inner wall and the attachment of the tympanic membrane posteriorly. Weber in bad ear. Schwabach lengthened. Rinne, right ear positive, about normal. Rinne, left ear negative, lengthened bone conduction, shortened air conduction. Whisper right ear 30 feet, left ear four feet. Immediate operation recommended, which was done following morning.

February 5, 1907, 11:30 a. m. Radical mastoid operation, closing by the Panse flap. Large pneumatic mastoid. The attic and antrum were filled with cholesteatomatous masses. The large and small mastoid cells as well as the cells about the zygoma were filled with cholesteatoma. There was but little pus at various places throughout the mastoid. All the cancellous bone was removed. The sinus was accidently uncovered. Operation completed.

During the afternoon and evening, patient complained considerably of pain on this side of the head and in the ear. Vomited several times. Temperature dropped to 99.6. The following morning temperature 100.8, pulse 106, respiration 104. Complaining of pain in the ear and on the whole side of

the head. Vomited several times. Evening temperature 100.4, pulse 96 and respiration 24. Second, third and fourth days cerebral symptoms presented increasing in severity from day to day. Complains of pain almost constantly, dizziness a great deal of the time. For two nights the pain was so severe that morphin was necessary to keep him quiet. The fourth day the right pupil more dilated than the left, reacts to light and accommodation. The papilla of the left eye more injected than the right. White blood count 20,000. Bacteriologic examination of pus in mastoid, streptococci. On the evening of the fourth day a second operation was done for purulent meningitis. On exposure of the dura it was found to be red and inflamed. The bone was removed until healthy dura was uncovered. The uncovered area was somewhat larger than a silver dollar. The dura bulged into the cavity made by the removal of bone and no pulsation was noted. The dura was incised and at the same time a meningeal vein was cut which gave considerable trouble in ligating, so as to what escaped I am unable to say. A culture was made which proved to be streptococci. After the hemorrhage was stopped the brain pulsation was quite normal, and the operation brought to a close. Salt infusion per rectum administered. The reason the brain was not incised was that it did not protrude and that there was no pulsation.

Vomited some during the night. Did not complain of pain during the night. Pupils normal in the morning.

Fifth day: Temperature 102.2, pulse 102, respiration 24. Complains of pain in the head occasionally. More rational than before operation. Temperature 100.8, pulse 72, respiration 18.

It will be noted that the temperature has increased rather than diminished. All this speaks of further infection and possibly more active because of the operative interference. From the fifth day to the eleventh, the cerebral symptoms continued to increase. Headache and pain on the side of the head almost constantly. Delirium increasing in severity from day to day, rational at times. Twitching of the muscles of both sides of the face, hands and arms. The following day paralysis of the hand and leg of the opposite side. Nystagmus. Cannot see at all. At intervals rational, when he complains of pain. This condition increased in severity, complete paralysis of both sides, complete blindness and no rational moments. Following

the second operation the cerebral symptoms were more pronounced. My explanation of this is that the meningeal infection became more aggravated, which is the usual course. This case illustrates very well what disastrous results follow in neglected cases. No doubt this boy had meningitis a long time before he was operated. His temperature never fell to normal. The lowest was 99.6, the morning following operation, increasing a few tenths every day with increasing cerebral symptoms. Had the infection taken place from the operated area the fever would not have appeared so soon. The same is true of all surgical affections. From a misunderstanding autopsy was not secured.

CASE 4. J. C. S., male, 75 years of age. Medical history by Dr. H. C. Moffit. Father and mother died in old age. Mother died at 70 from malaria. No severe sickness in family. Patient has always been well. No pneumonia or typhoid. Was in the army and had a sword wound over the left parietal. Has had malaria. Denies specific history. Five months ago had an acute suppurative otitis of the left ear. Has had headache over this side of the head ever since. At times it is very severe. The severe spells come every third day and are increasing in severity. Pain only on this side of the head. Memory failing. Hearing gone in left ear. Pain in left occiput, running down neck. Appetite is poor at times, due to nausea. Has no vomiting spells. Bowels regular, urine negative. Has noted laryngeal cough from fluid discharged in the pharynx. Has no dizziness. Has lost forty pounds in four months. Tenderness at back of neck when pain is felt. Has had to take morphin constantly for the last two months. No temperature nor chills. Examination: Pale, emaciated, evidently in much pain. Holds head stiff. Cannot bend it forward without much pain. Tenderness over the upper three vertebrae. A little swelling slightly to the left of the upper three vertebrae as well as back of the mastoid. Most tender between mastoid and vertebrae and on deep pressure is felt a swelling that feels partly periosteal and partly of the soft part. Along the anterior and the posterior borders of the mastoid are glands dwindling from above downward, the largest the size of a bean, somewhat tender. Pain along left jugular, but no tenderness. Pulse in jugular. Pupils small from morphin, but react to light and accommodation. No nystagmus. Fundus not examined. Skull not tender. No facial nor trigeminus

paresis. No change in reflexes. No swelling of vertebrae from the mouth. Examination of nasopharynx leads to a discharge of considerable quantity of pus.

Probable Diagnosis.—Extradural abscess; sarcoma in the posterior fossa; necrosis of the atlas or a sinus affection.

Ear Examination by Myself—The ear stands out from the temporal bone more than the ear from the opposite side. This ear is hot in comparison with the ear of the other side. Some swelling of the mastoid but particularly back of the mastoid. Sensitive over the whole of the mastoid, but especially back of mastoid. Some pain down the side of the neck. The pus is small in quantity and very offensive. There is decided bulging of the posterior superior wall. A small perforation in the tympanic membrane. Weber in bad ear. Schwabach lengthened in bad ear. Speech on contact in this ear.

Operation September 15, 1905. Radical operation. Started to do the operation for acute mastoiditis. However, I changed it into a radical operation because the pathologic findings were so extensive that I thought it could not be thoroughly removed without the latter procedure. Pus under the periosteum. The perforation was near the tip of the mastoid. On removal of bone, pus and granulation tissue welled into the cavity uncovered. The individual cells were largely destroyed and instead a large cavity was present. In curetting away the granulation tissue I suddenly encountered more pus under considerable tension. After further curettement I was able to demonstrate that this latter pus was in the sinus and separated from the pus in the mastoid by granulation tissue. I curetted the jugular end of the sinus as far as my curette would go and packed with iodoform gauze. The other end of the sinus was nicely sealed and the patient had no symptoms of pus infection, so the clot was not disturbed. The jugular was not ligated for the same reason. The only explanation that can be offered for this is that of a pneumococcus infection. Alexander, of Vienna, reports a similar case about two years ago, saying that he had made a thorough search of the literature and was unable to find a similar case on record.

The patient made an uninterrupted recovery. He did not have any chill, fever, or sweat during his entire illness.

Case 5. Male, age 32, machinist by occupation. Had ordinary diseases of childhood. Has never been ill so far as he can remember. On January 21, 1907, while slightly under the

influence of alcohol, he fell, striking the back of his head. Says that he was somewhat dazed for a time and noted that he had a discharge of blood from the right ear. The following day he noted a serous discharge from the ear which was tinged with blood. Also noted that he could not hear so well from this ear as formerly. Three days following the injury had a chill and some fever. Some pain in the ear and some pain in the mastoid region. This subsided gradually during the course of ten days, and he returned to work for a short time, when he again had pain and tenderness back of the ear. Stopped work for three or four days and then again felt quite well. This fever, pain and tenderness has continued interruptedly for the last six weeks. Three days following the accident, pus began to discharge from the ear and it has continued up to the present time and is of a very offensive odor. There has always been more or less pain confined to this side of the head. At times much exaggerated. Of late the pain is increasing in severity and occurring more frequently. For some days past says he has had fever. No chills or chilly sensations.

Ear Examination.—No swelling of the soft parts about the mastoid. No increased surface temperature. Slight tenderness over the whole of the temporal bone. Very sensitive over the tip of the mastoid. There is an offensive discharge from the small perforation below the end of the hammer. The tympanic membrane was bulging to such an extent that the landmarks were completely obliterated. The bulging of the posterior superior wall was so marked that it helped to obliterate the membrane. Temperature 102.5, pulse 110. Operation recommended.

As the following day was Sunday we did not operate until Monday, his temperature remaining near 103 the whole of the time.

Acute mastoid operation. Nothing of note on removal of the periosteum. After removing the outer shell of the mastoid a blood clot was found that was partly broken down and intermingled with pus. The clot began just back of the posterior osseous wall of the meatus, extending horizontally across the mastoid to the wall of the sigmoid sinus. On removal of the blood clot, granulation and pus, the fracture could be traced through the posterior osseous wall of the meatus, crossing the mastoid, fracturing and uplifting that part of the mastoid that covers the sinus; between the sinus and the

broken bone there was pus and new organized connective tissue covering part of the sinus wall. The sinus was uncovered until it appeared perfectly healthy. There was pulsation in the sinus and it was compressible. The balance of the mastoid cells and the cancellous tissue was removed and the antrum opened freely, and the operation completed.

The temperature chart did not drop as was expected. Wound dressed the second day following operation. No pus in the external meatus; everything looking well in the mastoid wound. The third day, the temperature remained about the same. I decided that the sinus could be explored the following morning. When I called on the fourth day the temperature had fallen considerably and the patient was feeling very comfortable. I decided to postpone further operative procedures. In less than half an hour following my visit, the patient had a chill and the temperature went up to 104. This temperature chart illustrates very beautifully the rise and fall of the temperature with the chills and the perspiration that followed. During the fifth day the patient began to experience pain in the right knee, which was bandaged and had hot applications applied. The following day, six days after first operation, I decided to open the sinus. The new granulation tissue was curetted away, all parts made clean as possible, pulsation could be felt in the sinus. Besides it was compressible. There was no pain along the jugular at any time. The lateral sinus was freely incised, the jugular end of the sinus did not bleed as much as the other end. At any rate, I concluded that there was a partial thrombus and I curetted and packed with iodoform gauze. The distal end was plugged without curettement. The jugular was ligated and the operation completed. The following day, patient had a chill and temperature of 104. The same day the knee was punctured and the secretion showed to contain a pure culture of streptococcus. The knee was freely opened the following day. There was considerable bloody pus found. It will be noted that there was a decided fall in temperature following this operation and another rise about two days following this knee operation. I account for this by the fact that the mastoid wound had not been dressed for three days. It will be noted that there was a gradual fall following this new dressing. Dr. Alvaris has done some bacteriologic work for me on this case, of which I wish to speak. Jarueth and others have recently been studying the polymor-

phonuclear neutrophiles and find that the number of neuclei vary considerably under different conditions and in different diseases. There are five classes of cells with 1, 2, 3, 4 and 5 neuclei respectively, the percentage of the classes and the average number of neuclei to a cell vary. Normally the count varies only slightly from the following:

I	II	III	IV	V	
5	35	41	17	2	Average number per cell 2.76.

The polymorphonuclear neutrophiles are supposed to develop from small myelonotic cell with single oval nucleus. A few of these are normally found in the blood. The older the cell presumably the more neuclei it has, and the older cells are supposed to be most active in the phagocytosis. If this be true a large percentage of multinucleate cells would give a good prognosis, as the person ought to be more resistant to infections. This seems to be borne out clinically but an immense amount of research must yet be done. A bad prognosis might have been given in Dr. Welty's case with pus and streptococci in the knee joints, but the differential neutrophile count showed the following percentages:

I	II	III	IV	V	
4	24	34	25	13	Average number 3.19.

As the average number seldom goes over three, the prognosis for this count was good. This was borne out subsequently. A blood culture was negative three days later. Probably the circulating streptococci had been destroyed very quickly.

2510 Washington Street.

LVII.

THE TECHNIC OF THE COMPLETE MASTOID OPERATION—IMPROVED, SHORTENED AND SIMPLIFIED, THROUGH THE DIGASTRIC ROUTE.*

By Wm. Sohier Bryant, A. M., M. D.,

New York.

The complete mastoid operation fulfills the requirements oftener and better than any other. It removes not only the macroscopically diseased bone and cells, but also those structures which show bacteriologic changes only when seen under the microscope. It is therefore the surest method of avoiding labyrinthine or sinus involvement or secondary operation, and is the one which I usually perform. The conventional incision through the skin and outer table of the mastoid process, followed by excavation of the antrum, is perfectly satisfactory when we do not intend *a priori* to remove the tip of the process and all of the cells, in spite of the fact that the knee of the sinus occasionally lies directly in the path of the operative procedure. But when it is previously decided to remove the tip, together with all the cells, I have found that the time of the operation may be shortened and its technic simplified, by attacking the bone, penetrating the outer cortex, and opening the process at the tip first.

I make the skin incision close to the posterior fold of the auricle beginning at a point on a level with the upper wall of the meatus. The incision is then carried in a curved line downwards and forwards to a point level with the tip of the process. The periosteum is next incised at the bottom of the wound on a line corresponding to the skin incision. Then, the periosteum is lifted carefully, beginning at the tip of the process and working upwards, extreme caution being taken that it be in even sheets. After the anterior and posterior periosteal flaps have been loosened, the periosteum is lifted from the mastoid process entirely, the operator working around under the tip and up to the bottom of the digastric.

*Read before the American Otological Society, 1907.

fossa. Next with a medium sized rongeur the tip is quickly removed, and then the outer table of the cellular area. The cells in the base of the process are now broken down with the rongeur and cleaned up with the curette, thus fully exposing the antrum.

The possibility of opening the sinus is thus rendered quite remote even when it is situated much farther forward than is normally the case. If the knee of the sinus encroaches upon the posterior wall of the meatus too closely to allow easy entrance to the antrum by the usual route, the latter can be readily approached from below.

If the process is pneumatic our best tool is the rongeur. It will accomplish the breaking down of the tip and the removal of the outer table and cell structure more quickly and better than any other. If the mastoid is sclerosed the tip is removed with the rongeur but time can be saved if the electric burr is used to do most of the remainder of the bone work.

Another advantage in the digastric route for opening the process lies in the fact that one can locate the position of the facial nerve at its exit from the stylomastoid foramen, because in uncovering the digastric fossa in the beginning of the operation, the stylomastoid foramen which is situated at the anterior or inner end of the groove is readily found.

Moreover, we find convalescence greatly facilitated and shortened by the preservation of the periosteum together with free incision of the drum membrane. Post-operative hearing is best if the attic is entered from behind and is not explored, especially if the convalescence chances to be rapid. Post-aural scars are reduced to a minimum by closure of the wound at the time of operation.

I have found that this technic consumes the least time consonant with good operative results and that the convalescence is all that can be desired in rapidity as well as in auditory and cosmetic efficiency.

LVIII.

A CASE OF MASTOIDITIS AND EPIDURAL ABSCESS; OPERATION AND RAPID RECOVERY.*

By W. Sohier Bryant, A. M., M. D.,

New York.

At the last meeting of this society there was some slight feeling of doubt upon the part of certain members whether one of the patients I showed was really alive or not. In order to remove this doubt and to assure these gentlemen that she is very much alive, I have taken the liberty of presenting a short history of the case.

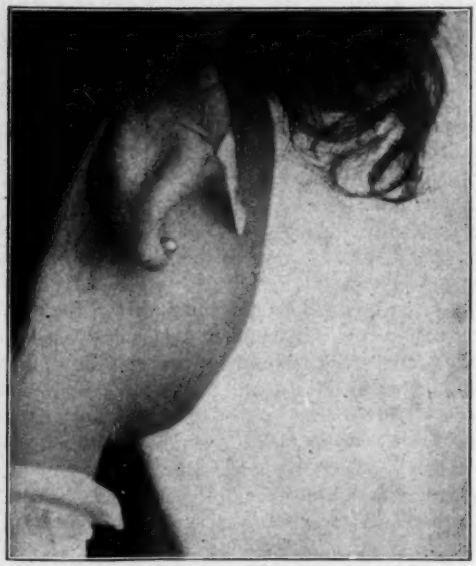
June 19th, 1906. Patient a girl of 16 years, well developed and well nourished, with partially occluded nasal fossae due to a deflected septum.

History.—Recurrent purulent otitis. For four weeks she has manifested very slight mastoid tenderness associated with a little pain. Inspection shows a very thick bulging nipple of the drum-membrane filling the canal.

Operation.—Advised and acceded to. A very free incision of the membrane was made. The skin was then incised behind the auricle down to the periosteum after my usual method. The periosteum was lifted from the mastoid. The bone felt rough but there was no abnormal amount of bleeding. When the cortex was perforated, about one dram of greenish pus welled out under pressure. The cortex was of medium thickness, the tip of the process and all the cellular structures were soft and full of granulations, and the surrounding bone was also soft and injected. The whole tip was removed and the sigmoid sinus was bared from the knee almost to the bulb. It appeared red, was sparsely dotted with granulations, and was soft and resilient to the touch. There were no occipital or epipetrosal cells and very few zygomatic cells. There was a perisinus abscess at the lower border of the knee of the sinus; the inner table was found absent over an area about as large as a dime. A small abscess was evacuated near the

*Read before the American Otological Society, May, 1907.

bulb posteriorly. All the cells were removed and the antrum opened; the antrum and attic were curetted. The dura mater was also exposed over the tegmen. Finally the posterior osseous wall of the meatus was taken down and the facial ridge curetted down to the facial nerve. The wound was washed with lukewarm normal saline solution which was allowed to remain in it. Blood for the blood-clot was supplied by rubbing the edge of the wound. A small cigarette drain



No. 1. Right Side. After Operation.

was passed from the antrum to the lower angle of the wound which was closed by a continuous subcutaneous silver wire suture and covered with gauze moistened in normal salt solution. Light packing in the canal. Rubber tissue was laid over all, then a layer of absorbent cotton held in place by a firmly applied bandage.

Convalescence.—The dressings were changed every day; the wet dressing was not re-applied.

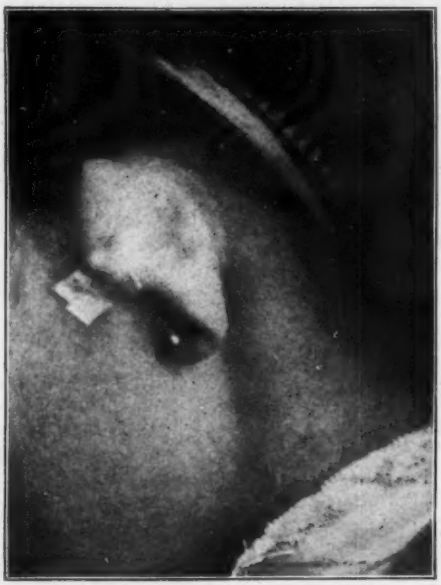
On the second day the outer dressings were dry and every-

thing looked well. The patient was out of bed and sat up in a chair.

On the third day the drain was removed and the patient remained up all day.

On the fourth day the meatus was found dry and the hole where the drain had been was closed. Boric powder was insufflated in the meatus.

The wire suture was removed without any pain on the fifth day.

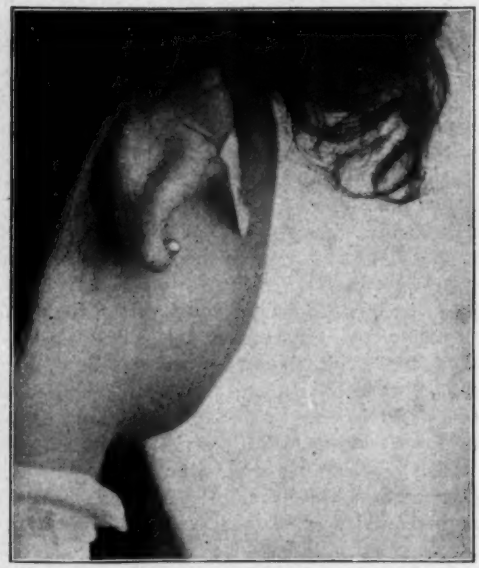


No. 2. Left Side. Not Operated Upon.

The patient went home on the sixth day. The course of the convalescence was so uneventful and rapid that one is led to the belief that the wound would have healed quicker without the drain.

On the seventh day dermatol and boric powder were insufflated on the drum membrane. The drum membrane had not yet healed; it was uneven, and allowed a trifling serous leakage. Boric powder was applied. The dressing and band-

bulb posteriorly. All the cells were removed and the antrum opened; the antrum and attic were curetted. The dura mater was also exposed over the tegmen. Finally the posterior osseous wall of the meatus was taken down and the facial ridge curetted down to the facial nerve. The wound was washed with lukewarm normal saline solution which was allowed to remain in it. Blood for the blood-clot was supplied by rubbing the edge of the wound. A small cigarette drain



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Convalescence.—The dressings were changed every day; the wet dressing was not re-applied.

On the second day the outer dressings were dry and every-

thing looked well. The patient was out of bed and sat up in a chair.

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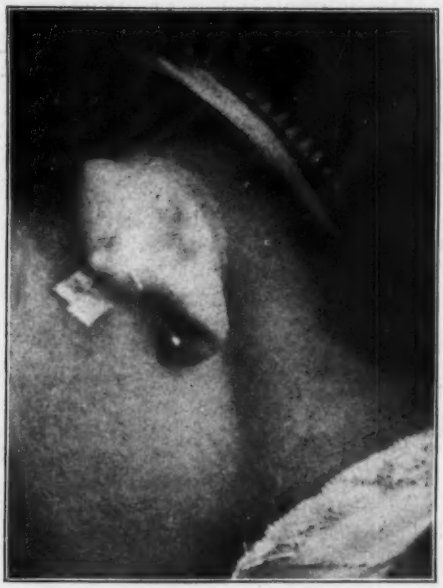

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On the seventh day dermatol and boric powder were insufflated on the drum membrane. The drum membrane had not yet healed; it was uneven, and allowed a trifling serous leakage. Boric powder was applied. The dressing and band-

age were left off and replaced by a small cotton cocoon. The watch was heard at 20 inches.

Post-Convalescent Notes.—On the eighth day all form of dressing was permanently discontinued and the patient was shown to the American Otological Society at the Academy of Medicine. The watch was heard at 18 inches, and the drum membrane was dry and pale.

The watch was heard at 36 inches on the ninth day.

On the eleventh day the watch was heard at four feet. The watch was heard at five feet on the fourteenth day. There was some tenderness and the tympanic membrane was thick and depressed.

All crusts had desquamated, and the scar was clean, linear and scarcely visible on the sixteenth day. The watch was heard at 5½ feet.

On the fifty-ninth day, the scar was linear and the surface flatter than upon the sound side. The watch was heard at 8½ feet. The drum membrane had cleared off well, but was still depressed and thick. Patient said she had had constant singing tinnitus since the ear began to discharge. Valsalva inflation negative. Politzerization positive. Examination of the nose and throat showed large tonsils and some adenoid tissue; the right middle turbinate was large and covered with muco-pus. The nasal septum was deflected to the affected side.

Seventy-first day. The post-aural surface had sunk a little, but it remained even. No regeneration of bone was apparent. The patient had sizzling tinnitus. The watch was heard at five feet. Hypertrophic rhinitis was marked and there were signs of postnasal catarrh. Valsalva inflation was positive and improved the hearing slightly, but did not move the malleus. It brought the posterior part of drum membrane a trifle nearer to view.

On the one hundred and twenty-eighth day the scar was scarcely perceptible. The drum membrane appeared normal, except for the opaque cicatrix in the posterior segment. The mastoid process seemed to have filled out somewhat since last examined. A watch was heard at five feet. She complained of frontal headaches. The turbinals were nodular and hypertrophic, and the mucous membrane was pale. Transillumination showed cloudy frontal and maxillary sinuses. The pharynx was dry.

Two hundred and forty-third day. Valsalva inflation nega-

tive. The watch was heard at 6½ feet. The tympanic membrane looked very well.

Three hundred and fifteenth day. No scar discernible on post-aural surface. The watch was heard at 11 feet in operated ear.

Summary.—The convalescence from the mastoiditis and epidural abscess was complete in six days; the operation wound healed by first intention in three days.

The convalescence from the middle ear was more prolonged, lasting seven days, with a final result of good hearing. The subsequent health of the patient has been an uneventful continuation of the aural conditions, which are good considering the nasal obstruction which was the primary cause of the disease. General health always excellent. The nasal condition was purposely not treated in order not to affect the post-operative processes of repair.

LIX.

A CASE OF SUPPURATION AND NECROSIS OF THE LABYRINTH—OPERATION—RECOVERY.

By George Loring Tobey, Jr., M. D.,

Aural Assistant Surgeon to the Massachusetts Charitable Eye and Ear Infirmary,

Boston.

August 15, 1907. M. P.; female; unmarried; 19 years of age. Seen in consultation for Dr. Croston, of Haverhill.

History.—At six weeks of age she had a right otitis media suppurativa; the ear continued to discharge for eight or nine months, when there was a succession of small post-aural abscesses, which were lanced by the family physician. The abscess eventually healed; the ear, however, continued to discharge, more or less, for four years, when she was seen by Dr. Clarence Blake, to whom I am indebted for the following record: "I saw M. P., four years of age, on March 17, 1893, on account of a suppurative process in the right ear with sequestrum formation on the posterior canal wall. This sequestrum measured six by eight millimeters in superficial area and extended to a depth of four millimeters into the cancellated structure of the bone. The operation consisted in its removal and the curetting of the resultant cavity down to firm tissue, with uneventful healing."

Following this operation, the ear was dry for six years. In the fall of 1899, following an attack of la grippe, the right ear began to discharge freely, no other aural symptoms being present.

The discharge continued during the winter, and early in the spring (1900) she was taken suddenly in school with severe headache, vertigo, nausea and vomiting; at this time she could walk with difficulty, but found that it was impossible to go up or down the stairs. She was confined to her bed for several days, owing to the nausea and headache; the vertigo was in no way influenced by the position of the patient; in so far as can be ascertained, there was no elevation of

temperature. The symptoms gradually disappeared and she was up and about within ten days. The hearing, which was impaired before, was not noticeably diminished by this attack.

Several weeks after this attack she discovered that when she attempted to lift anything or leaned far forward, she became very dizzy and would have to lie down for half an hour to an hour, at the end of which time she felt all right. This latter, together with a profuse discharge, continued with no other symptoms for two years. In the spring of 1902, following slight exertion, she was taken with very marked vertigo, nausea and headache referred especially to the occiput. The loss of equilibrium was so great that she was unable to stand without support, everything seemed to be revolving about her as the center. There was no temperature nor chills and the character or amount of the discharge changed in no way. The vertigo was present at all times and was in no way affected by position. There was at this time slight diminution in the hearing.

During the last six years similar attacks have occurred at intervals and the vertigo has been present to a greater or less degree since the very severe attack in 1902. The discharge from the middle ear has been constant, at times very foul and at other times mucoid in character.

Four or five months ago she noticed that the vertigo was more marked and that she was not quite sure of herself when walking. At this time she found in leaning over her bed that on reaching an angle of about 80 degrees her sense of equilibrium was lost. She then experienced a whirling sensation and fell forward on the bed. This occurred not once but invariably, and the patient tried it repeatedly. This whirling sensation was accompanied by slight nausea, which passed off in a few moments.

There was an increasing tendency upon the least exertion to a slight loss of equilibrium and a sensation as if the objects around her were moving in their own horizontal planes about her as a center.

Three weeks prior to August 19th she was taken with a severe chill lasting for twenty minutes, the temperature was not ascertained; very severe shooting pains radiated from the right ear toward the occiput; there was a fainting sensation with nausea and occasional vomiting. The loss of equilibrium was so marked as to confine the patient in bed;

there seemed to be an irresistible force causing her to whirl toward the right and at the same time she would fall, or, to describe it more accurately, collapse. All objects, even the floor and ceiling, seemed to be revolving to the right; that is, toward the affected ear. The whirling sensation was not affected by her position and was present with the eyes open or closed. The slight hearing which had remained in that ear was entirely lost. There have been no subjective noises in the ear within the patient's memory.

The very acute symptoms disappeared in six or seven days. The equilibrium improved somewhat so that the patient was able to walk with some guidance, the sense of elevation was almost entirely lost, so that she stubbed her toes and found great difficulty in going up or down stairs. The headache lost its shooting character, became dull and grinding, but was not definitely localized. The bewilderment of which she complained during the last year had become very much more marked and everything seemed confused.

As she walked into my office her gait and general attitude immediately suggested a case of cerebellar abscess, which we see not infrequently. She was a well-developed and, to all outward appearances, a very healthy girl. When she stood with her feet and knees together she could stand perfectly still, but as soon as she deviated from the position she lost her equilibrium and fell if not supported, everything seeming to whirl to the right, carrying her with it. There was slight nystagmus to the left. The finer coordinated movements were normal, as were the reflexes.

Posterior to the auricle and over the right mastoid bone there were several small cicatrices. There was no tenderness nor edema over the mastoid, nor were there any enlarged cervical glands. A very profuse creamy discharge, foul in odor, filled the right external auditory canal; when this had been removed the canal walls were found slightly congested, but with no apparent infiltration. The membrana tympani was absent, as also were the ossicles. The internal wall of the middle ear was covered with granulation tissue of considerable thickness. The outer wall of the epitympanum was necrotic. No bare bone could be felt over the promontory, but the probe was passed through the fenestra rotunda and its withdrawal was followed by the escape of a small amount of pus.

Operation.—August 19, 1907. Ether.

The usual mastoid incision was prolonged above in the line of the zygomatic process so that the auricle might be drawn downward and forward. The cortex was very rough and the periosteum was elevated with difficulty.

In doing the Stacke-Schwartze operation the bone was found to be sclerosed to a very marked degree, there being no cancellar tissue whatsoever in the mastoid portion. The antrum not being found at the usual depth and in the usual position, I decided to expose it by following along the posterior canal wall, this latter was removed by means of the rongeur and chisel. The space usually taken up by the antrum was found to be filled by firm ivory-like bone similar to that of the mastoid bone and contiguous with it. The facial ridge was shaved down as close as possible to the descending portion of the facial nerve. The horizontal semicircular canal was literally carved out of the firm bone surrounding it, there being no cancellar tissue surrounding the prominence as is usually the case. It was very evident that there was no erosion or necrosis of this canal, externally at least.

The outer wall of the epitympanum was removed to a level with the tegmen, granulations were removed from the epitympanum and the tegmen smoothed off with a curette; apparently, there was no exposure of the dura. The hyotympanum was also curetted and levelled with the floor of the canal.

The Eustachian tube was curetted, removing the granulations and mucous membrane. The whole cavity was packed with pledgets of cotton saturated with adrenalin-chlorid for a few minutes to control the hemorrhage from the granulations in the middle ear.

The bleeding having stopped, I was able to thoroughly examine the inner wall, which was covered with granulations. The stapes was not found, but the fenestra ovalis was filled with granulations, through which a small silver probe was readily passed into the vestibule; the withdrawal of this probe was followed by the escape of a small amount of pus.

The facial nerve, covered with granulations, was found exposed posteriorly and superiorly to the oval window, the intervening bone having been destroyed by necrosis, thus making the nerve one boundary of the niche; the anterior bony edge of the niche was soft and necrotic. The silver probe passed readily through the round window, the edges

of which were very soft and punky. The small bridge of bone between the fenestrae was *in situ* and was removed by means of a small gouged chisel. The bridge having been removed, the opening was enlarged by removing the outer wall of the vestibule and the lower portion of the promontory with the gouge (and curette), thus exposing the whole vestibule and the beginning of the first whorl of the cochlea.

The vestibule was completely filled with granulations and pus; these were removed with a small curette and the inner wall exposed, but no fistulae were discovered. The cochlea was cleaned out as far as exposed, but I did not care to risk further exploration of this part, owing to the danger of injury to the modiolus and internal auditory canal internally, the carotid artery anteriorly or the jugular below.

The horizontal semicircular canal was now further isolated by removing the bone well forward to the fallopian canal, the prominence was removed by means of a chisel, the applied force being in the plane of the canal and above the fallopian canal so as not to involve the nerve. The lumen of the canal on being exposed could be differentiated in no way from the firm white surrounding bone except by its outlines; there was apparently no fluid present nor were there granulations or blood. A small silver probe was passed through the canal and into the vestibule, pasing around the small bridge formed by the ductus fallopii. I did not feel justified in opening the other two canals, as I felt that if the horizontal was not involved in the necrosis, the other had in all probability escaped and that the infection was limited to the vestibule and cochlea.

The cavity was next swabbed out with alcohol. A Körner flap was made from the membranous and cartilaginous canal, the whole cavity was firmly packed with small pieces of iodoform gauze in such a manner as to hold the flap in position in apposition with the posterior wall of the mastoid cavity. The original incision was closed by interrupted silk-worm sutures.

There was a good recovery from ether.

August 20th. There was a great change in the mental condition of the patient; the utter bewilderment and confusion which had been present for two years or more had entirely disappeared. As she aptly expressed it, "It seems as though I were in another world where everything is quiet

and peaceful." The vertigo, which had been constant, was not present when she lay perfectly quiet, there was no headache and the nausea was no longer present. The loss of equilibrium, on the other hand, was more marked than before the operation, it being impossible even to sit up in the bed without support, the sensation being similar to that experienced in a small boat in a choppy sea, unattended, however, by nausea. Slight nystagmus to the left persisted. Considering the granulations on the facial nerve and the manipulations in their removal, it is surprising that there was no facial paresis. The outer dressings were changed and she was given ten grains of potassium bromid every four hours and liquid diet for twenty-four hours. She complained of no discomfort during the day and slept well the following night.

August 21st. The equilibrium was slightly improved, as she could raise herself to a sitting position and retain it without support; this exertion was accompanied by a whirling sensation, but there was no nausea. There was retention of the urine, requiring catheterization when required; otherwise, she was very comfortable.

August 23rd. The improvement of equilibrium continued and she was able to stand alone, but could walk but a few steps without support. There was no headache nor nausea and there was no return of the confusion present before the operation; the retention of the urine persisted till this morning. She sat up in bed with little or no discomfort.

August 26th. Patient was up and about the room and walked into the next room with no difficulty whatsoever; her equilibrium was practically normal, there was no vertigo whatsoever, there had been no headache nor nausea since the operation, one week before. She could lean far over and regain the vertical position without difficulty and without experiencing the whirling sensation of which she complained previously. The outer dressing was changed twice during the week, but the packing in the cavity was in no way disturbed. The temperature had not been above 99 degrees since the operation.

August 27th. I decided to risk a skin-graft, and on August 27th, one week after the original operation, the patient was again etherized. The packing was removed from the cavity, which was found to be perfectly clean, with granulation tis-

sue springing up throughout. The Körner flap was firmly adherent to the posterior wall of the mastoid cavity.

One large graft, two by three inches, was taken from the right thigh and this was placed in the ear on a plug of gauze. The posterior incision having healed by first intention, the sutures were removed.

August 31st. Since the skin-graft three days ago the patient has complained of no pain, nor has there been any rise in temperature, consequently the dressing has not been touched. This morning I removed the gauze plug, on which the graft had been inserted, and the cavity was perfectly clean, the graft apparently having taken. The cavity, having been carefully dried out by means of cotton pledgets, was left exposed to the air, the patient having no dressing whatsoever on the ear.

September 2nd. The cavity has been dried out twice daily, there being only a very slight moisture, the graft has apparently taken over the whole cavity. The patient is up and walking around, experiencing no discomfort whatsoever, there is no vertigo nor headache and her equilibrium is perfect.

The radical cavity was dried out daily for seven days, at the end of which time it was perfectly dry and the whole cavity completely epidermatized.

September 9th, twenty days after the original operation, the patient was discharged well, and told to report in ten days, which she did. The cavity was found in the same condition as when last seen.

To-day, four months after the operation, she is in perfect health, with the exception of total deafness of the right ear. There has been no return of the vertigo nor loss of equilibrium.

LX.

THE THERAPEUTIC VALUE OF FIBROLYSIN IN OTITIS MEDIA.*

By Ernst Urbantschitsch, M. D.

Translated by Clarence Loeb, A. M., M. D.

After Hans von Hebra showed in 1892 that thiosinamin is able to cure lupus and to soften pathologic scar tissue, this drug was accepted into our therapy and used to soften scars, at first in a few cases, urethral strictures (Hauc), in chronic inflammatory genital diseases (Latzko), in keloids, fibrous bands following varices, lepromata, syphilomata and lupus, in scleroderma (Juliusberg[1]), in adhesions (Lewandowsky[2]), in valvular insufficiencies following cicatricial changes, rhinoscleroma, Dupuytren's contractions, pyloric and esophageal stricture (Halasz[3]), glandular tumors (R. Kaufmann[4]), strictures, ankyloses and compressions, in the clearing up of clouded cornea, in synechiae of the iris, and even chronic neuritis and traumatic epilepsy. It was tried also in interstitial diseases of parenchymatous organs (hepatitis, nephritis, orchitis).

Thiosinamin has also been used in otology. Sinclair Tousey[5] reported in 1897 a case of very severe deafness which was considerably relieved by thiosinamin. Martin Sugar[6] obtains very satisfactory results by it in otitis media catarrhalis chronica (10 cases). L. Hirschland[7] used thiosinamin (and later fibrolysin also) in the same disease, as well as in affections of the nose and throat, and was very well satisfied with the results in some cases (the individual cases are not given in detail). These positive results are, however, opposed by negative ones, e. g., those of Bezold[8] and Vohsen[9].

The well known reaction of edema and softening of the tissue is at times macroscopically as well as microscopically evident. In a case of rhinopharyngeal lues, the fourth injection of thiosinamin was followed by an increased dyspnea in consequence of a severe swelling of the subglottic space, making a tracheotomy necessary.[10] Glas studied the histologic

*From the Monatschrift fur Ohrenheilkunde, XLVI, p. 63.

changes in the scar tissue after treatment with thiosinamin and found that the borders of the individual connective tissue fibres were much less distinct, their contour uncertain, the nuclei widely separated from one another, while the whole band seemed swollen and the fibres were stretched and puffed up.

"Microscopically as well as macroscopically,[11] we must consider the action of thiosinamin and fibrolysin as a serous transudation, which loosens up the hard inflammatory bands similarly to Bier's stasis treatment, softens pathologic products and makes them more suitable for resorption."

The reason that this remedy has not been used oftener lies in the fact that thiosinamin must be used by injection into the body, but since it is almost insoluble in water, it must be used in an alcoholic solution, which makes them very painful and therefore not always practicable.

It is, therefore, a very pleasant circumstance that F. Mendel[12] was able to prepare a substance, which he called fibrolysin and which is a double salt, composed of one molecule of thiosinamin and one-half a molecule of sodium salicylate. The important features about its physical properties are its solubility in water and its instability in the presence of light or air. Accordingly, the firm of E. Merck, Darmstadt, have introduced it to the trade in sterile, sealed, brown globules of 2.3 c. c. each (corresponding to 0.2 g. thiosinamin).

Since the beginning of March, 1906, I have made experiments to determine the value of fibrolysin in diseases of the ear. These were carried out in my private practice and at the ear department of the Allgemeine Poliklinik in Vienna (Professor Victor Urbantschitsch). I found, in the great majority of cases, a favorable action, so that I desire in this article to direct attention to the treatment of diseases of the ear by means of fibrolysin.

Since the use of this remedy per os is of so little value that it does not come into consideration, its employment is restricted to injections. These can be made in three ways: intravenous, intramuscular and subcutaneous. The first undoubtedly brings the quickest results, and should, therefore, be used when a very quick action is desired. It must be noted, however, that if the injection is not made in very wide veins, it may cause the formation of a thrombus by injury to the epithelium at the site of the injection. Therefore, only very

wide veins should be chosen, where a thrombus is rare. Since, however, treatment of ear diseases does not, as a rule, demand rapid action, and the whole procedure is a much more subtle one, this kind of injection can usually be disregarded.

The gluteal muscles are supposed to be the best place for intramuscular injection, as they are practically painless. Very few patients, however, permit this, partly on account of the discomfort of disrobing, and partly on psychic reasons. Furthermore this method is not so extraordinarily beneficient that it must be insisted on. There remains, therefore, as the most practical method the subcutaneous injections.

The site of the injection is theoretically of no importance, since the drug acts through the blood current, i. e., in every case is taken up by this and exerts its action from it. Nevertheless, a place near the focus of the disease is usually chosen with the hope that some beneficial local action might be exerted by the drug. For this reason, I made the injections, in the beginning, beneath the skin of the mastoid process, but soon found that the fluid was dissipated very badly in the subcutaneous connective tissue, on account of its poverty of fat. This resulted in a considerable tension of the skin over the fluid (of such amount that a quantity of ½ c. c. causes a swelling such as is found in acute mastoiditis following a perforation of the pus through the mastoid process under the skin) and furthermore it is not easy to introduce here an amount of about 2 c. c.. There is also the fact that this place is more exposed and therefore more easily infected if the plaster or collodion used to protect it falls off, and, furthermore, the deformity caused by the injection is very perceptible to one's fellows, to which many patients object. This soon caused me to give up this place and instead to make the injection into the arm, for which purpose the upper arm is better than the lower.

I make the injections usually in the upper arm, alternately on the right and left side, also in the thigh and the back. The sites of the injections are chosen as far apart from each other as possible, as those made near the place of some previous injection are occasionally painful. Of course, the injections are made under the strictest aseptic precautions.

At the first occasion, I inject only a small amount of fibrolysin, about 0.3 c. c., and if this causes no untoward symptoms, the dose is increased subsequently very quickly to 0.6

c. c., then to 1.2 c. c. and finally to an entire capsule of 2.3 c. c. I usually divide the latter dose into one-half for each arm, though most patients can stand the entire amount in one place. After the injection, I massage the place to aid in the absorption of the fluid.

The number of the injections naturally depends on the severity of the case. The average case requires from 20 to 30 injections, given two to three times a week. The indication is a cessation of the improvement, i. e., if I find in the course of the treatment that during twenty injections the hearing steadily if slowly increases, and then fails to do so during the three to five following ones, I usually stop giving them. If after eight to ten injections of the full amount no improvement has resulted, I do not continue them.

A point of special importance is the care of the syringe and needle after each injection. Since light causes fibrolysin to disintegrate into its component parts, the crystals are deposited in the plunger and in the needle, causing both to become brownish. Especially in using of syringes with ground glass or metal plungers which are unyielding and therefore cannot pass over slight inequalities of surfaces, this condition is very unpleasant. Therefore it is necessary to cleanse the needle and syringe after each injection with absolute alcohol. Furthermore, I fill the syringe with absolute alcohol until the next time. If through oversight the syringe or needle should become stopped up, the use of hot or boiling water will sometime remedy this.

It cannot be denied that the injections cause, in addition to the desired action, concomitant symptoms which exert an influence on the treatment, and are more frequent, according to my experiences, than they are reported in literature. These unpleasant complications can be divided into local and general.

The local consist of: (1) When the injection is hypodermic, there is a distinct burning sensation at the site of the injection, which usually, however, is very short (½ to 5 min.), though it may last several hours; (2) there is a yellowish blue discoloration of the skin around the site of the injection, which not infrequently is of considerable extent and may last for several weeks, gradually clearing up from the periphery to the center; (3) nodules may be formed at the site of the injection which frequently appear only after one to two weeks

and may become the size of a hazel nut. They are almost always painless, and disappear in the course of a few weeks or months. They are caused probably by a part of the fibrolysin being deposited under the skin, and the crystals causing (as a foreign body) an increase in connective tissue.

But the general symptoms which are occasionally caused by fibrolysin are without doubt more important, though they are absent in the majority of cases. The most frequent are more or less pronounced headache, congestion and dullness of the head, and general malaise. These may pass away in the course of a few hours, but in some cases they last a day or longer. In one case there was intense weariness with insomnia. The bodily malaise may be limited to the extremities, usually only one. In one case there was weakness in both feet and hands, in two only in the legs, in one only in the arms, in one only in the right hand, which at the same time was very cold. Nausea, also, was observed once. A very interesting feature of one case was the regular appearance of a menstrual hemorrhage about twelve hours after each injection. This artificial mensis resembled the normal in its course and duration (two to three days). For this reason I was finally compelled to discontinue the injections in this case, although they had exerted an unusually favorable influence on the ears. This phenomena was probably due to a hyperemia—in this case in the uterus—and is to be ascribed to an idiosyncrasy. As an analogue, hyperemia appeared elsewhere after each injection, as stated above, in the head, or, especially, in the nose, sometimes causing spontaneous hemorrhages. Perhaps the appearance of severe toothache, observed in one case after each injection, is to be explained by a hyperemia near a carious tooth.

In none of the cases with unpleasant symptoms did I use large doses. They appeared with injections of as small amounts as 0.1—1.0 c. c. It is not improbable that the use of full doses (2.3 c. c.) would not be accompanied by these symptoms, just as e. g., sedatives (veronal, etc.) in small doses excite the patient while large doses have a distinctly quieting effect. In most of the patients, these unpleasant symptoms appear only after the first injection, and do not reappear even after a lapse of several months between two injections, so that a certain tolerance can be acquired for the remedy, at least in this respect.

But just as unpleasant symptoms are manifested in connection with the injection, so also are favorable influences upon the general condition of the body. Many patients feel freer, especially in the head, and the bodily energy seems increased. In one case, there appeared an enormous appetite.

In the fibrolysin-cure, the injection is only one-half of the work. It must always be kept in mind that the action of fibrolysin is to soften and to increase the dilatability of cicatricial tissue. The latter must also be employed. An energetic local treatment must accompany the injection, directed against the chronic middle ear catarrh.

The local therapy consists in inflation, bougiering, vibration and friction—massage of the Eustachian tube, pneumomassage of the drum, massage of the ossicles by Lucae's pressure probe, massage of the ostium tubae, faradization and galvanization (katalytic action). It is best at each treatment to use some of these, and in some it is best to alternate them somewhat.

As to the choice of cases, in the beginning I used the cure in all kinds of severe deafness, cases of chronic middle ear catarrh, sclerosis, chronic inflammation of the middle ear with a dry perforation, and even labyrinth and nerve affections. This had the advantage of giving a view of the sphere of application of the remedy. To convince myself that the favorable action was actually due to the fibrolysin, I used it in cases which had been treated with the usual methods without results or at least with insufficient results.

My experiences show that the proper field for the use of the fibrolysin is the advanced chronic dry middle ear catarrh, adhesions in the middle ear, and the beginning stages of sclerosis, especially when the hearing is not constantly bad, but has occasional increase in amount. In the severe forms of sclerosis I was unable to accomplish anything objectively, by 10 injections at least, and so discontinued the cure, partially because I considered that in this case there was already an ossification present which could hardly be influenced by a remedy against cicatrization, and partially on account of the restricted amount of fibrolysin at my command, which I could not waste on almost hopeles cases. Furthermore, the cases of disease of the inner ear resulted negatively. I did not, however, use more than 10 injections. I will not assert, nevertheless, that even in such doubtful cases, a longer treatment

would not accomplish an improvement. I saw even the hearing remaining after a otitis media purulenta chronica antiqua considerable increased.

Fibrolysin has an occasional undoubted influence on the subjective noises even in those cases where there is no considerable loss of hearing. This is the case when they are due to an irritation or slight compression of the nerve fibrillae of the acusticus, which are relieved by fibrolysin. This is analogous to the drug's favorable action in neuralgia. There is relief from tinnitus also in the cases of deafness which are relieved or influenced by fibrolysin. On the other hand, it sometimes happens that the subjective noises remain although the hearing increases, but the favorable action usually affects both. I consider fibrolysin contraindicated in old people with arteriosclerosis on account of its property of causing congestion. It is not advised in active or recently cured otorrhea, also, as this can be increased or started up again.

The prognosis at the present can, of course, not be made, even probably. It is to be hoped that deafness caused by disease of the sound conducting apparatus can be considerably relieved.

Fibrolysin, therefore, is a remedy by which we can achieve results not possible in any other way, and its reception into the otologic therapy can be hailed only with joy. However, we must not regard it as a panacea for deafness. Each man must learn the suitable cases, and its proper employment. This article is to help show the correct way and to act as a standard to aid in its employment. Many may find its use harder than it seems.

In order to illustrate better the use and action of fibrolysin, I will report a number of cases treated with it:

Case 1. Mrs. W., 50 years, very advanced otitis media catarrhalis after otitis media purulenta subacuta. Hearing for forks and clock 0. Fibrolysin injection (0.2 c. c.) 3/6/06. In the course of the day, there appeared severe headache and staggering gait, causing the patient to fear other injections, which were therefore not continued.

Case 2. Mr. K., 39, letter carrier. Watch not heard at either ear; very severe tinnitus, left more than right. First fibrolysin injection 3/6/06 (0.5), toothache such as never before. Burning at site injection for three hours. Second injection 3/9 (0.8); severe toothache in afternoon, less tinnitus.

Eighth injection 3/23 (2.0), tinnitus at times less, hearing unchanged. Unfortunately the treatment had to be discontinued on account of private circumstances of the patient.

Case 3. Mrs. M. W., 46. Sclerosis, very severe bilateral tinnitus, clock 0. First injection 3/7/06 (0.5), followed by decreased tinnitus during the day. After the seventh injection (2.0), the tinnitus remained minimal until the next time (one week later), hearing unchanged. After the eighth, the patient experienced a distinct improvement, since she could plainly hear the rattle of her wagon. The fifteenth injection was made on 6/16. The result was a purer, better hearing for loud sounds, the clock was not heard, and the tinnitus had disappeared on the right side while it was very slight on the left. When the patient presented herself on 6/25/06 and 1/2/07, there had been no decrease in this result.

Case 4. Mrs. M., 45 years. O. M. C. C., watch right heard ad concham, left not. First injection 3/10/06 (0.8). In the afternoon she had "hot head," which she felt after second and third injections also. On account of severe headache after fourth injection, patient asked to be excused from further injections.

Case 5. Mr. F. P., 45, laborer. Labyrinth affection. Watch right 0, left 6/150. Rinne positive bilaterally, Weber not lateralized, Meniere's symptom complex. The treatment was desired especially on account of the latter. First injection 3/12 (0.4), second 3/14 (0.8). After the injections, the patient felt much lighter in his head, and, also, remained free from attacks until 3/20, although formerly there were two attacks weekly. On the 3/20, there was a very severe Meniere's attack, which lasted the whole day. For this reason I discontinued the injections.

Case 6. Miss Th. H., 22. Cat. chron., watch right 6/150, left 6/150. First injection 3/15 (0.9), second 3/19 (1.5). Thereupon headache for two days. Third injection 3/21. An hour later there again appeared a severe headache lasting until the morning of 3/23. For this reason, treatment was stopped.

Case 7. Miss R. S., 24. Cat. chron. with tinnitus. Watch right ad conch., left 2/150. First injection 3/16/06 (1.0), second 3/18 (1.5), third 3/21 (2.0). Watch, 3/23, right 1/50, left 17/150. Whisper right 6 m., left 1 m. 3/25 watch right 5/150, left 22/150. Eleventh injection 4/18; watch

right 9/150, left 27/150, tinnitus varying, in general better. Sixteenth injection 5/6, watch right 9/150, left 30/150. One hour after first injection, patient experienced bitter taste in whole mouth, apparently also on the lips, then sensation of heat in head, "as in fever" (two hours later), then malaise of the entire body; the arms seemed heavier, the gait was weary, but the tinnitus was distinctly less. These symptoms lasted 2 to 4 hours. Afterwards, the head was much freer, hearing and sight better. The subjective noises at first remained the same, but later became distinctly less. The improvement in hearing and tinnitus continued to the beginning of July, 1906. As the result of catching cold while on a steamer, a relapse occurred. Accordingly, I made on 7/16 an injection of 2.0 c. c. fibrolysin. Two days later, the hearing was again distinctly better. There was a "much freer feeling in the head," the tinnitus decreased to the point of disappearance, and this favorable condition lasted throughout the entire summer until 9/20/06, when the patient had a severe attack of influenza which caused another relapse in the condition of the ear.

Case 8. Miss L., 26. Cat. chron., with subjective noises (right a tone, left a noise). Watch, right 30/150, left 15/150. First injection 3/27/06 (1.0); eighth on 4/12. Watch, right 45/150, left 40/150. Tenth injection 5/1. Watch, right 56/150, left 24/150. Tinnitus less, the right tone much weaker, and the left noise now two tones, one strong high and one weak, deep tone, which the patient considered much less unpleasant than the previous noise.

Case 9. Mr. H. H., 50, laborer. Sclerosis. Watch both sides 0; whisper, right 30 cm., left 20 cm. First fibrolysin injection 5/6/06 (0.5); fourth 5/19 (0.6). The treatment had to be discontinued on account of severe weakness of feet, pain in lions and headache after the injections.

Case 10. Miss F. R., 20. Rinne bilaterally positive, Weber indeterminate. Deep tuning forks not heard, middle and high much shortened. Watch, right 8/150, left 15/150. Whisper, right 230 cm., left 400 cm. Tinnitus. First injection 5/7/06 (0.5). Local burning for one hour, right hand tired and cold. About noon sleepy; slept for a quarter of an hour (never slept before in the afternoon); no headache, but severe, general weariness. For rest of the day entirely well. Second injection 5/9 (0.3). Local burning one-fourth hour, then both arms, especially right "enormously tired;" severe dull

headache in afternoon. Went to bed earlier, slept badly, awoke with slight headache. Left side, tinnitus gone, although it had commenced 6 years before and had been continual for 3 years. Third injection 5/14/06 (0.15). In the morning, headache for 3 hours, malaise. Patient wished to sleep but could not. By 4 p. m. entirely well. Fifth injection 5/18 (0.2). Headache scarcely one-half hour, no malaise, tinnitus less. On 5/19, patient had attack of angina lacunaris, from which she frequently suffered, and which always had taken at least a week for recovery, but which this time left her in 3 days. Ninth injection 5/28 (0.4). Tinnitus distinctly less; patient cannot remember when she felt so well in her ears. Weariness of the feet (following preceding injection into buttock). Eleventh injection 6/1 (0.5). Very tired, trembling of feet, sleepiness with inability to sleep. Patient suddenly called home for family reasons.

Case 12. Miss M. H. Cat. chron. with tinnitus. First injection 6/1/06 (0.2). Afterwards severe tinnitus for 2 hours, then much less. Headache in afternoon, especially over right eyebrow. Third injection 6/7 (0.4). Tinnitus and hearing better; headache, nausea, vomiting, heavy feeling in abdomen. "Taste of iron in mouth," peculiar feeling in nose. Discontinuance of injections on account of unpleasant concomitant symptoms.

Case 13. Mrs. L. L., 36, supernumerary in royal opera. In May, 1906, severe deafness followed a bad case of grippe, accompanied by tinnitus left ear. Watch both sides 0. Whisper, right 100 cm.; left 45 cm. Speech, right 360 cm., left 90 cm. Rinne negative bilaterally, Weber indeterminate. First injection 10/15/06 (0.5), no concomitant symptoms. Fifth injection 10/24 (2.0), hearing distinctly better. Patient does not have to pay such strict attention to test, while on the stage understands whispers and piano which has not been possible since May, 1906, in spite of constant treatment. Tinnitus much less, absent for hours at a time (formerly continuous). Eighth injection 10/31 (2.0). Watch, right ad concham, left 0. Whisper, right 100-200 cm., left 60 cm. Speech 800 cm. right, left 200 cm. In the opera house, patient hears piano and piccicato, which she had not heard 14 days 14 days before. 2/22/07, watch, right ad concham, left 0. Whisper, right 8 m., left 6 m.

Case 14. Miss F. C., sister of preceding patient. Two sis-

ters of her mother also deaf. Beginning sclerosis, tinnitus four years, "ringing bells" right, for two years left. Since then continually under treatment, without result. Hearing steadily diminishing. Rinne negative bilaterally. Weber lateralized towards the right. Watch, right 0, left ad concham. Whisper, right 35 cm., left 300 cm. Speech, right 80 cm., left 600 cm. First injection 11/30 (0.5). Fifth 11/12 (2.0). Twenty-third 1/2/07. Watch, right ad concham (?), left 1 cm. Whisper, right 550 cm., left 650 cm. Speech, right 600 cm., left 700 cm. Tinnitus much less.

CASE 15. Miss G. L., 32 years, sclerosis. Watch, right 3/150, left 1/150. First injection 5/22/06 (0.1). Ninth injection 6/19 (1.0). Twelfth 6/30 (2.3). Sixteenth 7/13 (2.3). No special concomitant symptoms. Hearing distinctly better. Watch, right 7/150, left 4/150. The improvement lasted through the summer until 10/9/06. In consequence of severe cold and influenza, a decrease in hearing resulted.

CASE 16. Mr. F. F., architect, 38. Beginning sclerosis. Watch, right 3/150, left 70/150. First injection 10/29/06 (0.5); fifth 11/7 (2.0). Watch, right 10/150, left 100/150. Sixteenth 12/7. Watch, right 20/150, left 80/150. The patient, for years, when walking with a person, could go only on the right side of his companion if he wished to hear what was being said, but now can walk on either side. The improvement in hearing for speech, which is even more distinct than the watch, of course makes his life more pleasant.

CASE 17. Mr. A. B., officer, 42, sclerosis. Watch not heard at either ear. First injection 1/24/07, no unpleasant concomitant symptoms, appetite increased, freer feeling in the head. Ninth injection 2/11; hearing for speech and music distinctly better, for the watch unchanged. The appetite has increased to an amount never known before. Thirteenth injection 2/20; tinnitus, for the first time in years, was absent for several hours, the hearing has increased so much that the patient for the first time since the summer of 1905 could clearly distinguish every voice of a sextette at a musicale. Patient will be treated further.

In each of these cases, the fibrolysin treatment was preceded by several months of local treatment which achieved little or no result. It is especially interesting that in many cases the hearing was very considerably increased for speech, tone and sound, if not for the watch.

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13. This firm kindly sent me gratis a large number of globules for use in the Allgemeine Poliklinik.

ABSTRACTS FROM CURRENT OTOLOGIC, RHINOLOGIC AND LARNYGOLOGIC LITERATURE.

I.—EAR.

A Case of Cerebellar Abscess and Sequestration of the Labyrinth, Due to Chronic Middle Ear Inflammation.

W. SCHUTTER (*Tydschr. v. Geneesk.*, July 28, 1906) saw on February 3, an 18-year-old girl, who had a running right ear for about ten years, which troubled only so far as the hearing was concerned; severe headache with vomiting and dizziness and tendency to fall toward the diseased side had lately appeared, and a week after the commencement of these attacks facial paralysis on the diseased side. Schutter found the surroundings of the right ear normal, bone not painful on pressure, Romberg with tendency to fall toward the right; the visual field turned toward the right, movements of the eyes and pupils normal, pulse 85, the external ear canal filled with granulations and pus. Admitted to the hospital two days later. Entire examination negative, temperature 38. Patient is deaf for speech; tuning-fork examination unreliable. The facial paralysis may be due to a lesion in the labyrinth or in the tympanum; the headache, dizziness and vomitus could also be due to labyrinthitis. Radical operation (February 19); nothing particular was found, especially no fistulae; first dressing after six days for slight elevation of temperature; thereafter every other day; appetite, general appearance, good. March 6, sixteen days after the operation, patient began to complain of headache. Increase of intermittent headache, mostly in the forehead, sometimes in the back. She screamed with pain, was dizzy with the sensation of falling out of the bed towards the diseased side, nausea, sometimes vomiting and slight delirium. When she lay down after having set up a while, she said she got a short, severe pain in forehead and occiput. No pain of the skull on percussion, but paresis of the right abducens and speech defect; dysarthria without any trouble in the motion of the tongue or other articulatory muscles; in both eyes neuroretinitis hemorrhagica; no convulsions. For one day the pulse was 60, otherwise no diminished frequency. On March 11 a lax paresis of the right arm, also ataxia of the right leg,

but less pronounced than of the right arm. The tendon and skin reflexes were normal, as well as the sensibility and localization for dermal stimuli. The only result of tuning-fork examination is that C is heard osteo-tympanally somewhat shorter on the right side than the left. A complication in the posterior part of the cranial cavity is most probable, as the hemiparesis, ataxia and dysarthria point to it; most probably there is a cerebellar abscess, as no symptoms of meningitis are present. Operation on March 12: tegmen tympani and antrum carefully looked over again, no fistula visible, then the genu superius of the sigmoid sinus and a part of its vertical part was uncovered; sinus looked normal and pulsates, medially the dura looks normal but does not pulsate; on further exploration the posterior wall of the antrum is found sequestrated, with the sequestrum touching the dura. Puncture here revealed a somewhat muddy fluid, but no pus; tamponade with iodoform gauze. For a few days patient felt better, but then the headache returned now constantly in the forehead with dizziness and vomiting. One day there was severe neuralgia in the collateral trigeminus and sometimes spasms appeared on that side of the face. Fetid discharge was found in the tympanum on changing the dressing (March 16). That same day trepanation was performed over the right temporal lobe; the dura did not pulsate, looked normal; a probe 3 to 4 cm. deep into the cerebral mass freed some serous fluid, after which the dura pulsates. The cerebellum was again punctured and fetid pus drawn out of the abscess; opening was enlarged and a drain put in, through which a large quantity of pus is discharged. Pulse before operation 100, after 80. It did not do patient much good; headache and vomiting persist. Exitus on March 31st. The dysarthria and right abducens paresis had increased during the last days, the knee reflex became abolished while the plantar reflex remained; autopsy showed dura adherent to the posterior surface of the temporal bone around the fistula. Toward the cerebellar abscess, nearly the entire hemisphere changed into an abscess and the inferior horn of the right cerebral ventricle enlarged, and in the os petrosum a large loose sequestrum containing the vestibulum with the semicircular canals and a part of the cochlea.

The dysarthria as a cerebellar symptom is fully in accordance with Bolk and Jelgersma. Bolk localizes the coördi-

nated movements of the muscles of the head and neck in the anterior part of the middle portion of the cerebellum, on evolutionary data; while Jelgersma found in cases of cerebellar atrophy with dysarthria the cerebro-cerebellar tract on both sides injured. Jelgersma showed that these parts (Bolk) belong to a cerebro-cerebellar tract, which can be demonstrated through the whole series of mammals and the development of which goes equally with the power of continual finer differentiated coördinated movements of the muscles of the head, a differentiation which reaches its highest point in the movements of speech and mimicry in men. An apparent exception is made by a focus in the pons, but this must be very near the raphe and enough extended to affect both the tracts.

The dysarthria in this case was due to a monolateral but very extensive abscess in the direct neighborhood of the vermis, the sagittal dimension of which was a good 3.5 cm. The possibility of double interruption of the cerebro-cerebellar tracts seems therefore very acceptable, and an encephalitic malacia around the abscess of the left hemisphere in the neighborhood of the abscess is probable, although without microscopical change. *Blaauw.*

The Itard-Cholewa Symptom and Its Detection in Cases of Sclerosis Aurium.

H. ZWAARDEMAKER (*Tydschr. v. Geneesk.*, July 28, 1906) includes the cases which the Germans consider to depend upon primary osteitis of the labyrinth capsule and those which result from a dry chronic middle ear catarrh. Even if one accepts the original pathologic-anatomic distinction, a differential diagnosis will be only possible in the later stages. And even here it is uncertain. Not only diagnostically, but also etiologically, these two conditions meet (family disposition, parasyphilitic and paratuberculous predisposition, influence of catching cold and trauma). They form one clinical picture, which can be taken as an entity for therapeutic purposes. Acoustically a disappearance of the bass side of the scale is found (Bezold); outspoken paracusis Willisiana; at first increase, later small decrease of the upper limit (only for the Galton whistle of Koenig). The writer adds a new symptom, the tactile insensibility of the drum for air motions en masse. It is easily shown by examining all cases with a tuning-fork of 32 vibrations. With sufficiently large movements, which pro-

duce considerable amplitude of vibrations, the observer notices a peculiar vibration in the region of the drum, which is the more striking as the sound is not more heard. The sufferer with sclerosis lacks this sensation when the whispering voice is heard at less than two meters. This phenomenon is identical with the symptom of anesthesia of the drum described by Itard and Cholewa. This is more positive as it is generally combined with lessened sensitiveness of tragus pressure and penetration of air on catheterization. It may be connected with dryness and scaling of the skin of the external auditory canal, so often described.

This same symptom is also found in extensive cicatrices of the drum and in hysteria. Sufferers with labyrinthitis and with neuritis acustica feel the vibration of the drum very well by motion en masse, even if the acustic acuity is far less than whisper at two meters.

STRUYCKEN divided sclerosis patients into two groups: those with diminished sensibility of drum, auditory canal and auricle and those with normal sensibility. He found the minimum limit of vibrations for contact as sensitive perception about 1 micron, while this went down to 5 to 6 micron in a number of cases. *Blaauw.*

The After-Treatment of the Radical Operation.

A. C. H. MOLL (*Tydsch. v. Geneesk.*, July 28, 1906) mentions the importance of the after-treatment in preventing recurrences. In the beginning the aim was to keep the form of the operation cavity as perfect as possible; continued tamponing was necessary, which, however, irritated, so that often epidermization followed only after much scraping and cauterizing. This made many surgeons desist from tamponing. Moll thinks everything is in favor of leaving the wound quiet. If the operation has made a septic wound, then after removal of the first bandage the cavity will be filled with sanguinous serous fluid with bactericidal properties. Piffl examined this fluid and found that in the succeeding dressings the strepto-, staphylo-, and diplococci decreased in number and virulence and ceased to act pyogenic. If the wound cavity is protected against infection from without, if the granulations are not irritated, then the cavity becomes slowly smaller and takes the form of the external earduct with an enlargement deeply inward, through addition of the attic and prominence of the promontorium. However, sometimes we must stimulate e. g. with tincture of iodin

or cauterize with chromium or nitrate of silver. As a rule, after the first bandage the evacuation of the secretion has only to be looked after by a moist piece of gauze lying at the bottom of the ear canal, which is in contact with the dressing which fills the external ear. This opening is enlarged at the operation, but no plastic, no flap is made. He therefore makes two parallel incisions from within outwardly in the posterior wall of the membraneous ear canal before opening the antrum and cuts this skin flap at the concha. This is the method of Zaufal. Many French surgeons remove the membranous posterior wall entirely, which facilitates the after-treatment very much, enlarges the cavity, and especially with children. Moll makes the upward incision according to Siebenmann, through the ear canal outwardly as far as the rima helicis through skin and cartilage, dissects the skin loose, clips the cartilage away with a downward and forward curve and lays the loosened skin over this cartilage defect behind, through which the deep parts come into sight. The wound behind the ear is closed; the cavity tamponed, which fixes the flap of Siebenmann at the same time. After removal of the first dressing no more tamponing. Where there is much secretion, daily change of dressings.

Many cases undoubtedly will remain where tamponing will be necessary, in general where for some reason the retro-auricular opening is not closed primarily. But whenever this is possible a reduction of the cavity should be tried and the healing process as little as possible be disturbed. He has not seen any unpleasant stenosis; he remembers two children operated years ago where the ear canal remained very ample. He mentions a case operated without removal of the membranous posterior wall in a child where repeated stenosis occurred which only stopped with removal of the posterior wall. Moll removes this wall now from principle, as this skin possesses mostly small vitality and the flap is in the way more or less for the after-treatment. Also during the operation itself, especially in children, a larger cavity has its advantage. He never found the hearing suffered with this method. *Blaauw.*

A Case of Post-scarlatinous Bilateral Osteomyelitis of the Temporal Bone, with Sequestration of a Part of the Left Labyrinth.

P. TH. L. KAN (*Tydsch. v. Geneesk.*, July 28, 1906) operated radically on a girl of 3 years who, during an attack of scarlatina,

acquired a double otorrhea, and eleven weeks later an abscess behind both ears. Nearly the entire mastoid process on the right side was removed in four loose sequestra; dura mater and sinus became bare after the operation and facial paralysis appeared, later relieved. On the left side the entire mastoid process was found movable, surrounded with pus. The mastoid process formed with a part of the lateral wall of the facial canal and the largest part of the horizontal semicircular canal one large sequestrum, which could be removed. The facial nerve remained intact. The hearing acuity could not be taken with the young child. During the healing the child learned to walk, and no disturbances could be noticed in the movements. The child now walks normally.

Report of Three Cases of Infective Sinus Thrombosis.

RICHARDS, New York (*Archives of Otology*, vol. xxxvi., No. 4). Case 1. A woman was stricken with la grippe and three days later a left acute otitis media developed, with mastoid pain. She became profoundly septic and on operation the antrum contained a few granulations, but no pus. The sinus was exposed and opened. From the torcular end a free flow occurred, but from the bulb end of the sinus there was no flow whatever. The wound was packed and a jugular resection done. Upon returning to the mastoid wound to remove the clot from the deep limb of the sinus, when the gauze packing over the lower end of the opened vessel was withdrawn, a recent thrombus was extruded and this was followed by a profuse gush of blood from the bulb. On the following day there was another rigor, but from this time on convalescence was uninterrupted.

Case 2. A middle-aged woman, who five weeks previously had a right acute otitis media purulenta, with mastoid tenderness and labyrinthine disturbance.

The middle ear inflammation abated, but the labyrinthine symptoms became greatly aggravated, with repeated vomiting, disturbed equilibrium and intense high-pitched tinnitus.

On operation no pus was found in the mastoid, but there were numerous necrotic areas scattered throughout the bone. The stapes had disappeared and the oval window was the seat of a fistula. The patient's condition improved to such an extent that she was allowed to return home. She was suddenly seized with a chill and, on account of inability to otherwise account for this condition, the vertical sinus limb

was opened and found to contain a purulent clot which extended into the bulb. On resecting the jugular the vein and its tributaries were so generally thrombosed that no clamping of vessels was necessary. The vein was cut off behind the clavicle and a curette passed far downward into the thoracic portion of the vessel, but no return flow was established. In a few days the patient succumbed to general sepsis.

Case 3. A child aged 11, with a long-standing suppuration of the left middle ear, developed mastoid tenderness, became delirious and had an irregularly high temperature. The neck in the upper jugular region was tender and decidedly stiff. There was no vomiting, no incoordination and the equilibrium was good.

On operation the antrum was found filled with thin fetid pus, there was an epidural abscess over the middle of the vertical sinus limb and a dark red clot with purulent foci extended from the knee of the sinus into the bulb.

After operation the horizontal nystagmus, which had persisted up to that time, ceased, and the further history of the case was uneventful. *Campbell.*

The Demonstration of Disturbances of Equilibrium in One-Sided Disease of the Labyrinth.

KROTOSCHINER, Breslau (*Archives of Otology*, Vol. xxxvi., No. 4). The most reliable tests for making a diagnosis of one-sided disturbance of the labyrinth are as follows:

1. Hearing tests. It should be determined whether on the diseased side forks from a' down are not perceived (Bezold), and the Lucae-Dennert test should be applied.

2. Static examination on a horizontal plane. The patients stand on the right leg, on the left leg and on their toes with closed eyes. The trunk is bent to the right, to the left, forward and backward.

3. Dynamic examination (walking and jumping). The disturbances of equilibrium are most evident usually on jumping backward with eyes closed.

4. In examination with the goniometer the degree of inclination which is supported with the eyes closed is always distinctly less than when the eyes are open.

5. Centrifuging:

(a) Active: Where the patient turns ten times to the

right about his own axis, and then to the left, with open and with closed eyes,

(b) Passive: Where a rotating disk five feet in diameter is employed. Five rotations are made, each taking five seconds. If the centrifuge is suddenly stopped there is a sensation of counter-rotation which persists for a few seconds. According to von Stein, in a normal person there is no counter-rotation. *Campbell.*

Herpetic Inflammations of the Geniculate Ganglion.—A New Syndrome and Its Aural Complications.

HUNT, New York (*Archives of Otology*, Vol. xxxvi., No. 4). The syndrome is dependent upon a specific herpetic inflammation of the geniculate ganglion, situated on the facial nerve in the depths of the internal auditory canal, in the entrance to the aqueduct of Fallopius, and its expression is found in herpes of the tympanum, auditory canal, the concha, tragus and antitragus. Because of its proximity to the facial nerve and the terminations of the auditory nerve neural complications are not infrequent; peripheral facial palsy, tinnitus aurium, deafness and symptoms of Méniere's disease. *Campbell.*

A Case of Serous Meningo-Encephalitis, with Autopsy Report.

BLAU, Goerlitz (*Archives of Otology*, Vol. xxxvi., No. 4). A child aged 2¾ years was taken ill with measles and three weeks later a right acute suppurative otitis media developed, with anorexia, malaise, gradual loss of eyesight and hearing, left-sided ptosis and convulsions in the left arm and the left leg.

Three weeks later the author found the child somnolent, emaciated, with reflexes absent and abdomen retracted. The left pupil was dilated and irresponsive. The right Mt. was almost completely gone, and the tympanum was granular and contained fetid pus.

The antrum was opened and its mucous membrane found swollen and granular. The middle fossa was exposed and the dura found tense and congested. The brain was punctured in three directions without finding any pus, though a large quantity of clear fluid was evacuated on each insertion of the knife. Exposure of the sigmoid sinus and posterior fossa gave the same result.

On autopsy the longitudinal sinus contained coagulated

blood. The pia was transparent and its vessels very thin. the convolutions were unusually flattened. Both lateral ventricles were enormously dilated and the thalami and the corpora striata flattened. There was much watery fluid.

Campbell.

Another Case of Otitic Purulent Sinus Thrombosis without Fever.

SCHROEDER, Erlangen (*Archives of Otology*, Vol. xxxvi., No. 4). A man, aged 49, five months before coming under observation had a left otitis media suppurativa with mastoid pain, which still persists.

On examination the canal was found filled with fetid pus, the upper wall depressed, the right red and bulging and edema and tenderness over the mastoid. The pus contained staphylococci.

On operation the antrum contained pus. When the sinus was exposed it was found covered with discolored granulations and pus exuded from the sinus through an opening large enough to admit a thumb. The whole course of the disease was afebrile. The sinus wound was packed with gauze and the clot above and below left undisturbed. *Campbell.*

Report of a Case of Diphtheria, Complicated by Acute Purulent Otitis Media, Mastoiditis and Infective Sinus Thrombosis.

KERRISON, New York (*Archives of Otology*, Vol. xxxvi., No. 4). A woman aged 19, suffering from diphtheria, had a temperature varying from 98° F. to 104.8° F. On the sixth day a paracentesis of the right Mt. was done because of redness and bulging. Mastoiditis developed and the antrum and mastoid cells from tip to zygoma were explored. The vertical limb of the sigmoid sinus was exposed for about five-eighths of an inch, but it appeared normal. For eight days following this operation the temperature was normal, then there was an abrupt rise to 105° F. An examination of the wound revealed a pus pocket, which was freely drained; still the patient became progressively more septic. The sinus was exposed from knee to bulb, opened, and while there was free bleeding from above there was none from below. A curette dislodged a well-organized clot about one-third of an inch long. The jugular vein was excised and recovery thereafter was uneventful. *Campbell.*

Otitic Meningitis.

ARNOLD KNAPP, New York (*Archives of Otology*, Vol. xxxvi., No. 4). Recently it has been shown that uncomplicated otitic meningitis occurs as often after acute as after chronic purulent otitis. The meninges are first infected, in nearly three-fourths of the cases, in the posterior cranial fossa and in slightly over one-fourth of the cases in the middle cranial fossa.

Heine suggests the classification of purulent meningitis into encapsulated, acute progressive and general. No single symptom is characteristic. Kernig's sign is perhaps the most constant. Lumbar puncture is a great aid. Until recent years the prognosis was most unfavorable. Then cases were reported as cured by elimination of the primary focus and by repeated lumbar puncture. *Campbell.*

II.—NOSE.

Concerning the Bleeding Polypi of the Nasal Septum.

JORGEN MOLLER (*Archiv. fur Laryngologie und Rhinologie*, Bd. xx, Heft 1, 1907). The first to describe these peculiar tumors was Lange. Schadewaldt first gave them the name of bleeding polypi of the septum.

These growths, in the large majority of the cases, occur in women, and in many cases there appears to be a distinct connection between pregnancy and the development of such growths. In an interesting case reported by Wright, the growth recurred several times after removal from the nose of a pregnant woman. In another year, when the patient was again pregnant, the growth developed again and recurred after removal. It finally disappeared spontaneously after the woman was confined.

Traumatism is given as an etiological factor by some writers (Freudenthal and Glas), while Glas also thinks that rhinitis anterior sicca is an important cause.

He made an accurate histologic examination in ten cases, and in seven was able to determine the presence of rhinitis anterior sicca.

The author reports two typical cases of this interesting condition. In both cases there was a distinct relationship to pregnancy. *Theisen.*

Contribution to the Treatment of the Purulent Inflammation of the Frontal Sinus.

H. J. L. STRUYCKEN (*Tydschr. v. Geneesk.*, July 28, 1906) makes, under local or chlorethyl anesthesia, an incision of 1-2 to 3-4 cm. below the inner upper margin of the orbit under the brow through the skin as far as the bone, not longer than 1 cm., and pushes away the periosteum with one of the small raspatoria of Killian. In the incision a self-retaining retractor is inserted and the inferior wall of the frontal sinus is perforated with Doyen's burr (1-2 cm. diameter) in the direction of the middle of the horizontal brow-line. Most of the hemorrhage stops when the refractor is put in place, sometimes adrenalin tampons are necessary, or a few artery forceps. Only a branch of the supratrochlear nerve has to be cut. When the bone-opening is large enough the membrane in view is carefully examined, and if the wall of the cavity is filled with pus it is easily perforated with the blunt probe. The pus flows out and the cavity is probed. When the bony wall is intact, the cavity is syringed with Anel's syringe, taking care that the fluid can escape at the side of the canule; or the pus can be sucked with Bier's suction bag, or both methods may be combined in different positions of the head. In acute and subacute cases the frontal headache, fever, etc., disappear immediately and the patient is cured without further effort. In chronic cases, where the nasofrontal duct is more or less obstructed, the anterior part of the middle turbinate is first resected, polyps, etc., removed. Complicating empyema of the antrum, primary or secondary, is relieved by way of the nose or alveolar process. The ethmoidal cells, if affected, are attacked endonasally. While syringing the nasofrontal duct is seen to be well open during the first few days, but later the fluid escapes along the external opening. As a rule in chronic cases one must syringe for a long time; to make it easy and painless a small drainage tube is put in or a tight wound silver spiral 1 cm. in length. They must be constructed so that they keep themselves in the wound and can be easily closed. It is remarkable how, with regular irrigation twice to four times a day with some warm non-irritating fluid, the condition in the nose changes, how not only the secretion of the frontal cavity changes its nature, but also the fetid character of the ethmoid secretion disappears and the purulent secretion diminishes. Marked hyperplastic mu-

cous membrane in the frontal sinus retrogrades within a few weeks. In one case a very fetid chronic multiple empyema of years' standing was cured after four weeks with a hardly perceptible cicatrix. However, one must be careful not to declare the condition cured too soon; the wound might need reopening, which then can easily be done with the pointed probe of Weber.

This method can not entirely replace the radical one, but it may be tried first; in any case it makes conditions more favorable and has undoubtedly great use. *Blaauw.*

Hyperthropic Nasal Catarrh and Complications with Clinical Illustrations.

BUCKLIN, New York (*Archives of Otology*, Vol. xxxvi., No. 4). The author claims that the vacuum formed within the entire respiratory tract with each forcible nasal inspiration amounts in cases of patients suffering from hypertrophic rhinitis to 1-3600 pounds to the square inch, and with the obstruction reduced one-half the amount of oxygen inspired is doubled.

He demonstrates these atmospheric relations by means of his "respirometer," a glass tube four feet long with an one-eighth inch bore. One end of this tube is placed in a glass of water, the other end is held air-tight in the patient's mouth, while he takes a long, rapid, forcible inspiration through the nose. The height to which the water is raised in the tube determines how great a vacuum the patient forms in the respiratory tract with each forcible inspiration. Each nostril is closed in turn, and it will be seen that inspiration through the more occluded nostril will cause the water to rise higher in the tube. *Campbell.*

An Epidemic Pneumococcic Catarrhal Disease.

BECK AND STOKES (*Journal of the American Medical Association*, September 14, 1907). A description of a curious catarrhal condition of an epidemic nature occurring in Dr. Beck's practice in Baltimore.

It differed essentially from ordinary colds in that it was apparently not the result of exposure and it affected almost invariably more than one member in a family, and sometimes entire families.

The cases may be looked on clinically as measles without a rash, as conjunctivitis with general catarrhal symptoms, as whooping cough without a whoop or as grippe without men-

tal or physical depression. The group of cases presented a definite clinical picture, and the authors considered that the epidemic is an affection having a distinct entity.

The paper is based on the study of fifty-six cases, of which thirty-three were examined bacteriologically.

The histories of a few of the most typical family groups are given:

Group 1. G. family, consisting of six members—father, mother and four children. The children were aged 18, 6 and 5 years and 10 months, respectively. The author was called to see the baby, who was thought to be suffering from whooping cough. The child had had none of the acute infectious diseases. The one boy, aged 5 years, was the first one in the family affected with this catarrhal condition. The others, except the other boy, were affected three days later. The attacks were ushered in with chills, followed by slight fever and night sweats; this was associated with paroxysmal cough, discharge from the nose, watering of the eyes, slight soreness in the throat, slight pain in chest and abdomen and absolute loss of appetite. There was marked constipation and vomiting frequently occurred after paroxysms of coughing. The cough was of a severe character. The attacks occurred more frequently during the night.

When the child was first seen the symptoms had subsided, except the cough and the discharge from the nose, which was then muco-purulent. He had a slight bronchitis and vesicles of chickenpox.

The one son, aged 6 years, who was similarly affected was also taken with chills and fever, which were followed by sweats. He had peculiar dry, paroxysmal cough, having three or four severe attacks at night, which lasted until he vomited. He had marked lachrymation, without congestion, nasal discharge, sore throat, thoracic and abdominal pains but no rash. The fever lasted about one week, during which he had several night sweats.

The daughter, aged 18 years, and the mother had the same train of symptoms, namely, fever, sweat, cough, sneezing, rhinitis, mild conjunctivitis, pain in the chest, slight abdominal cramps, anorexia and vomiting after coughing. The symptoms lasted three weeks. The father had the same symptoms, but a more intense conjunctivitis.

The baby began with intestinal symptoms, nausea, vomit-

ing and diarrhea. She had a muco-purulent conjunctivitis in both eyes, slight swelling of the eyelids and intense photophobia. The throat was uniformly red, but the tonsils were not enlarged. Cover-slip preparations and cultures from the nose and throat of the mother and baby showed pneumococci.

Group 2. S. family. Four members were affected—aunt, aged 35 years; mother, aged 40 years, and two children, aged 5 and 7 years. Mrs. M. and her sister, living in a flat on the second floor of the same dwelling, were also affected. The symptoms in this group of cases were about the same as in Group 1.

One of the most interesting cases of this collection was treated in the Baltimore Eye, Ear and Throat Hospital.

The patient, a boy aged 18 months, had had early symptoms of hoarseness, cough, fever and lassitude. Several brothers and sisters were affected in the same way and some of them had a rash. The eye symptoms had existed a week before he was admitted to the hospital. There was cloudiness of the cornea of the right eye and a hazy appearance of the cornea of the left eye. The mucous membrane of the mouth and throat was reddened and there was a slight, dirty looking membrane which appeared in patches in the pharynx. Cultures proved negative for diphtheria, but bacteriologic examination of the eyes showed almost pure pneumococci.

Etiology.—That this disease is of an infectious nature is evident from the report of the two family groups. Forty-six cases occurred in nineteen families.

An interesting example of the epidemic character is an outbreak on board a bay steamer, in which 20 per cent of the crew suffered the same symptoms, including the captain, chief engineer and watchman. A smear from the engineer's throat showed epithelial cells containing as many as sixty pairs of encapsulated diplococci.

In all except ten of the authors' cases there was a history of house infection.

Age.—The disease is met with more frequently in children. In twenty-two of the fifty-six cases it occurred under 7 years of age, ten of these under 1 year, the youngest patients being twins 8 weeks old.

All of the authors' cases occurred in the spring.

Symptomatology.—The period of incubation ranges from two to seven days.

The invasion is usually marked by chilliness, slight febrile disturbances, and occasionally night sweats. Temperature usually from 99.5 F. to 102 F., continues from three to four days to one week.

A spasmodic cough, croupy in character, usually worse at night and often associated with nausea and vomiting is a fairly constant feature.

Profuse herpetic eruptions frequently occur. Loss of appetite and often complete anorexia are distressing symptoms in young children. A striking feature in the study of these cases is the absence of any marked nervous or mental symptoms.

A decided leucocytosis was observed in all cases in which the blood was examined.

A significant feature of this disease is the formation of a pseudo-membrane. This occurred in 12.5 per cent of all the cases. The membrane is of a light yellow color, usually situated in the nose, pharynx or naso-pharynx, and occasionally the conjunctiva.

Cover-slip preparations show an enormous amount of encapsulated pneumococci, and direct inoculation with animals produces pneumococci septicemia. Severe bronchitis was present in ten of the cases. Some of them, which had been carefully studied, proved almost beyond a doubt to be due to a pneumococcus infection.

Fibrinous pleurisy complicated two cases, and frontal sinusitis and purulent otitis media each complicated one case.

Bacteriologic study.—In most of the cases the diplococcus pneumoniae, at times in combination with the pyogenic micrococci, was found.

The first cases of pneumococcic conjunctivitis were reported by Parinaud and Morax, in 1894, and the cases were also accompanied by lachrymation, coryza and purulent and fibrinous inflammation of the conjunctival mucous membrane.

So far the writers have studied 33 cases, and found pus cells and pneomococci in 7 specimens from the nose, 9 from the throat and 10 from the eye. The pneumococcus was found in cultures in 8 cases from the nose and in 23 cases from the throat and in 4 of which membranes were present.

The original cultures from most of the cases were inoculated into white mice, and the pneumococci often proved virulent, killing the mice with general septicemia in from 1 to 11 days. In the second series of 16 cases, 8 rabbits were

inoculated intravenously with pure cultures from the throats and 5 died of general pneumococci septicemia. In 29 out of 33 cases the pneumococcus was obtained either from the nose, throat or eye by cover-slip cultures or animal experiments.

Conclusions.—This disease exists as a distinct entity characterized by purulent or fibrinous inflammation of the mucous membranes of the eye, nose and throat.

The infection can be communicated from diseased to healthy persons.

The infection is caused by the pneumococcus, resembling in its cultural and pathogenic properties the cause of lobar pneumonia. *Theisen.*

III.—MISCELLANEOUS.

A Pebble in the Left Superlobar Bronchus, Extracted With Help of Inferior Bronchoscopy.

Dr. P. Th. L. Kan (*Tydschr. v. Geneesk,* Aug. 25, 1906) was called June 25th, 1906, to see a child of nearly 3 years, which had swallowed a pebble on the previous day while playing; symptoms of severe suffocation were present. According to the physician the pebble was loose, but could not be removed. There was first slight dyspnea and some hours later such a severe attack of dyspnea, that they feared he would choke, but before tracheotomy could be performed the oppression ceased spontaneously. Examination directly after the first and second attack showed the respiratory sounds over the left superior lobe practically absent; no other changes. No shadow with a Roentgenograph. On account of the age and nature of the foreign body, superior bronchoscopy was inadvisable; as the trachea of a child of 2 years has a diameter of 7 to 8 m. m., which necessitates the thinnest tube of Killian (7 m. m.), which is still too big to be moved in the bronchus. Therefore Dr. Pastoors performed tracheotomy. Careful palpation with a probe distinctly revealed a hard foreign body in the left main bronchus at a distance of 5.5 c. m. from the inferior angle of the tracheal wound and the probe went downward in certain directions without touching the body. Kan concluded from this that the pebble was tight in one of the branches of the chief bronchus and projected partly in this. This branch was in all probability the bronchus of the superior lobe, as the respiratory sounds were absent here. The shortest and thinnest bronchoscope of Killian could only with dif-

ficulty be pushed as far as the bifurcation, although the mucous membrane had been cocainized and the tube rubbed with sterilized oil. A small wall of mucous membrane was pushed up. It was impossible to see clearly into the chief bronchus and detect the pebble. Dilatation did not give a sufficient result. Then the nose speculum of Hartmann with very small and long branches was introduced; as it was 3.5 c. m. long it would remain at a distance of 2 c. m. from the foreign body, but this speculum somewhat stretched the trachea. The pebble now was seen and a repeated examination showed that there was considerable room between the pebble and the medial wall of the bronchus. A thin blunt sound was bent 2 to 3 mm. at right angle from the end, while a similar bending was made at the other end as a guide. The child was deeply narcotized, with hanging head; after a few trials the extraction succeeded. The entire operation, inclusive of the tracheotomy, lasted a full hour. The pebble had an oval form, was yellow, 1 cm. long, 7 mm. broad, 5 mm. thick.

In this case the diameter of the trachea was exceptionally small, probably smaller than the left main bronchus; perhaps the bronchi are more dilatable, having less cartilage than the trachea.

After extraction, a tracheal canule was introduced; during the night the respiration became difficult and in the morning the child seemed in danger of dying; oxygn inhalations improved things somewhat, but only the removal of dry, sticky mucus from the trachea after removal of the tube and dry crusts of mucus deep in the trachea improved the respiration; the child could then expectorate. The tracheal wound now was closed; continuous steaming in the neighborhood of the child, entire recovery. *Blaauw.*

The Treatment of Stuttering.

W. Posthumus Meyes (*Tydschr. v. Geneesk.*, July 28, 1906) mentions that psychical influence has its significance in heredity, and that stuttering and backwardness together with adenoids suggest degeneration. At present four methods are used: 1. The classical method of Gutzmann, conscious exercising of respiratory, speech and articulatory muscles toward one harmonic coöperation. 2. The method of Liebmann, the

suggestive or psychologic method; he combats chiefly the fear of speaking; only one person at the time is treated. Slowly self-confidence returns. 3. The method of Chervin, unjustly called after his pupil Berquand, enforces silence outside of the lessons, so that the impulse for spasmodic, extra movements of the face is suppressed. All three methods need five to six weeks for a cure, with constant control to prevent recurrence. 4. The combined whispering and respiratory method takes some principles from each of the three preceding ones.

As the causa morbi is so often a different one, no single method can be called the only true one. The chief point of the treatment is a good instructor, who succeeds in getting the pupils under his personal influence. Every disturbance of normal nose breathing must be treated. Stammerers can be benefited with extra instruction in speaking after the usual school hours. Stutterers are to be taken to a special institution where they are taught how to speak in addition to the usual instruction. *Blaauw.*

Neurologic Observations Regarding Stuttering.

Prof. K. Heilbronner (*Tydschr. v. Geneesk.*, July 28, 1906) states that the acquired stuttering conditions which the neurologist sees belong to different groups: 1. The aphasic stuttering (Pick, etc.); nearly related thereto, different forms of speech-disturbance following epileptic fit, permanent (Fuerstner) or transitory. 2. Those forms which develop from mutism, the post-traumatic stuttering. Heilbronner sees an analogy in this last form where stuttering starts suddenly after a fright; nearly related thereto is congenital imitation. Analogous to the first mentioned form, he considers the cases lately described, and also observed by himself, where facial-lingual pareses exist dependent upon a circumscribed cerebral disturbance. Some cases of left-handedness with stuttering belong to this group also. Chorea is closely related to this form. A lesion in the motoric apparatus somewhere must be sought for as the cause of the first group of stuttering; for the second group the lesion must lie outside of this apparatus. Etiologically a separation might be made between congenital disturbances dependent on local conditions, upon heredity or intrautering lesions (hypoplasias, as Pfister explains enuresis), and general disturbances, which produce diminished strength and thus a predisposition to all

possible psychical derangements. One must also think of the possibility of postpartum lesions (encephalitis). Prognostically it must be considered whether or not many obstinate cases of stuttering in non-neuropathic persons are due to such focal affections.

Therapeutically the method of education by conscious physiological speech is alone rational when a disturbance of the motor apparatus is found. For the psychogenous group this method has in reality the significance of a suggestive remedy. The success of the other methods shows that the suggestion is the principal factor. The stuttering treatment is a therapeutical question, which ought to be regulated by the physician. He has to decide if local changes need treatment. The eventually necessary exercise treatment, which must be individual and not schematic, may be left to the pedagogue.

Blaauw.

Speech Motions of Stutterers.

F. H. Quix (*Tydsch. v. Geneesk.*, July 28, 1906) examined several stutterers with Zwaardemaker's instrument for registering the speech motions. The following are registered: 1, the movements of the lower jaw; 2, the motions of the upper lip; 3, of the lower lip; 4, the movements of the muscles of the floor of the mouth. Together the time is registered and, if desired, the respiration. The curves must be compared with those of normal persons. The curves of normal persons are always uniform at a certain position of the instrument for the same word. The height as well as the succession of the curves, depend upon the intensity and the velocity of the diction and of the accent. There is always a constant connection between the moments of beginning of the motion of the different muscle groups. In the pause all muscles rest, only with accidental swallowing, the curve of the floor of the mouth shows a pretty steep elevation. The curves of stutterers show that one or more muscle groups do not rest between the pronunciation of words. Often in all muscle groups involuntary contractions appear, sometimes also only in some. Only in a few cases were these movements absent; they were generally strongly represented.

The muscular movements on pronunciation of a word show various departures from the normal type, which, however, may be found often in stutterers. The simplest case is that in which the stuttering spoken word shows a repetition of the

strokes in the curve. The size of the different strokes may greatly differ. The size gradually increases till at the end the right strokes appear. The curves become more complicated when the strokes of one muscle group increase and those of others decrease. Gradually a curve-image forms, where one muscle group makes repeated contractions in trying to pronounce a word, while one or more, the contraction of which is also necessary for that same word, remain quiet. With such a speech-motion the word can not appear before the mutually correct coördination is brought about.

In a normal curve the contractions of the different muscle groups begin at mutually constant times; with the stutterer, often one or more muscle groups move while the others remain behind. This must interfere with the normal explosion. Different anomalies are often mixed.

The speech motions of stutterers are characterized by: 1, contractions in the pauses between the speaking of words; 2, repetition of the typical contractions of the different muscle groups, with varying intensity; 3, variability of the moments of beginning of the contractions of the different coöperating muscle groups; 4, variability of the mutual relations of the intensity of these contractions. A coördination disturbance is suspected, and Quix thinks that the cause must be looked for centrally in the motor-speech center. It is a question if these anomalies are found in all stutterers; a more extensive investigation is therefore necessary. *Blaauw.*

Tracheal Diphtheria with Recurring Formation of Pseudo-Membrane.

H. Herzog (*Deutsche Medicinische Wochenschrift*, No. 20, 1907). The author had occasion to observe three times in a severe case of diphtheria "ecouvillonnage" of the larynx.

In 1896 Variot and Bayeux described, under the term "ecouvillonnage," a new procedure in the treatment of laryngeal diphtheria.

The method consists in the passing of the tube, just as in the ordinary intubation. The tube, which loosens the membrane clinging to the walls of the larynx and trachea, is removed in a few minutes and the detached membrane is coughed out.

In the following case described by the author the patient coughed out the tube with a membranous cast of the trachea three times.

J. B.; aged 2¾ years. Had measles four weeks before coming under the author's observation. Coryza and a hoarse cough for a week, with suffocative attacks for several nights. On examination an extensive membrane was observed on both tonsils. The nose was clear. Intubation was performed within a half hour after the patient was admitted to the hospital.

The tube could be easily introduced, but was coughed out after a few seconds, covered with a membrane which covered it like the finger of a glove.

The membrane showed the impressions of the tracheal rings.

Ten hours after the first intubation urgent symptoms demanded a second one.

The tube was again coughed out at once, and was covered with the same kind of membrane.

Fourteen hours later another intubation had to be performed, and the same thing happened. The same tube-like membrane was coughed out.

The further history of the case is of no great interest. The patient made a complete recovery.

The use of antitoxin did not prevent the regeneration of the membrane in the trachea, which occurred twice in twenty-five hours, but the ease with which the membrane was thrown out was undoubtedly due to the action of the serum.

Theisen.

BOOK REVIEWS.

A Text-Book of Diseases of the Nose and Throat.

By D. Braden Kyle, A. M., M. D., Professor of Laryngology and Rhinology, Jefferson Medical College; Consulting Laryngologist, Rhinologist and Otologist, St. Agnes Hospital; Fellow of the American Laryngological Association, etc. With 219 illustrations, 26 of them in colors. Fourth edition, thoroughly revised and enlarged. W. B. Saunders Company, Philadelphia and London. Price, $4.00.

The appearance of the fourth edition of Kyle's textbook emphasizes the position which it has held since the first edition was presented. The general plan is the same, but it has been greatly amplified in the revision.

The following new topics have been added:

Taking cold, lithemic rhinitis, chemic ulcers, fibromyxoma of the nasopharynx, glioma of the nose, telangiectoma, syphilis of the septum, empyema of the antrum in the young, bone-cysts of the accessory sinus, rhinopharyngitis mutilans, gangrene of the tonsil, actinomycosis of the tonsil, glandular pharyngitis lateralis, Vincent's angina, angina ulcerosa benigna, cyanotic pharyngitis, angioneurotic edema, pharyngeal aneurysm cough, purpura hemorrhagica, congenital s[illegible], scleroma of the larynx, bronchoscopy; voice, speech, defects of speech and relation of voice to hearing, functional aphonia and surgery of the larynx.

In the following chapters alterations and additions have been made: Simple, acute rhinitis; phlegmonous rhinitis; occupation rhinitis; simple chronic rhinitis; emphysema of the face, nasal obstruction and the importance of nasal breathing; ozena, atrophic rhinitis; nasal hydrorrhea; nasal syphilis; lupus; nasal neuroses; asthma; epidemic influenza; headache; neuralgia; erythema; epistaxis; tumors; fibroma of the larynx; lipoma of the tonsil; carcinoma of the larynx; sarcoma of the nose, fauces, pillars and soft palate; adenocarcinoma; cysts; deformities of the septum; tumors of the septum; angioma of the septum; depression of nasal cartilage; diseases of the accessory sinus; empyema of the sphenoidal and frontal sinuses; relation pathologic conditions of the nose, and accessory sinus to the eye; systemic nasopharyngitis; simple chronic nasopharyngitis; adhesions of the soft palate to the pharyngeal wall; diseases of the tonsils; diseases of the pharynx; hyperplastic change in the pharyngeal structure; rheumatic pharyngitis; syphilis of the pharynx; retropharyngeal abscess; pharyngomycosis; foreign bodies in the pharynx;

malformations and deformities of the larynx, edematous laryngitis; hyperplastic laryngitis; singer's nodules; syphilis of the larynx; tuberculosis of the larynx; prolapse of the laryngeal ventricle; hysterical aphonia; and laryngectomy.

The author details his well-known views on sialosemiology from the standpoint of the diagnostic value of saliva. The significance of the bacteria of the nose also receives attention.

In considering the various diseases of the nose and throat a most successful attempt is made to classify them in a logical way. As an example showing the character of this effort, the classification of acute rhinitis may be instanced, as follows:

I. Simple acute rhinitis.
   a. Acute rhinitis in constitutional conditions.
      1. Measles.
      2. Pertussis, or whooping-cough.
      3. Scarlet fever.
      4. Smallpox.
      5. Typhoid fever.
      6. Rheumatism.
      7. Diabetes mellitus.
      8. Diphtheria.
      9. Erysipelas.
      10. Scorbutic rhinitis.
      11. Anemic rhinitis.
      12. Scrofulous rhinitis (strumous).
      13. Caseous rhinitis.
      14. Epidemic influenza.
      15. Lithemic rhinitis.
   b. In the young.

II. Membranous rhinitis.
      1. Croupous or pseudomembranous.
      2. Fibrinoplastic.
      3. Diphtheritic (see diphtheria).
   c. Occupation rhinitis (traumatic).
   d. Hyperesthetic rhinitis (hay fever).
   e. Ulcerative rhinitis.
   f. Edematous rhinitis (acute edema).
   g. Phlegmonous rhinitis.

In like manner, all the conditions of the nose and throat are taken up and described in a logical, clear and painstaking way.

The typography is particularly commendable, the illustra-

tions well selected and clear, and the general index is far superior to what is found in most books.

Tracheo-Bronchoscopy, Esophagoscopy and Gastroscopy.

By Chevalier Jackson, M. D., Laryngologist to the Western Pennsylvania Hospital, the Eye and Ear Hospital and the Montefiore Hospital. With five colored plates and many illustrations. The Laryngoscope Co., St. Louis. Price, $4.00.

Jackson has been so closely associated with the progress of our knowledge of the direct inspection of the larynx, trachea, bronchi, esophagus, and stomach, that the publication of his work will be accepted as its authoritative expression. That this is justified is easy to ascertain by a study of his book.

It differs materially from the monographs of Gottstein and Schroetter, which are more exhaustive in the discussion of the individual topics but less clear and comprehensive.

He describes and pictures the various head lamps that may be used, such as Kirstein's and Guisez's, and, in turn, the examination and operative instruments of Mosher, Mikulicz, Rosenheim, von Hacker, Starck, Killian, von Schrötter, Einhorn, Ingals, Coolidge. The instruments which the author devised are described in detail. Those intended for examination differ from Killian's in that they carry a small lamp in the tube for the purpose of illumination, doing away entirely with the cumbersome head lamp.

The illustrations showing diagramatically the position of of the patient, assistant operator, etc., are particularly happy, and make the method exceedingly clear. A full discussion of foreign bodies in the treachea and bronchi make this part of the work most important, and the cases reported add greatly to the knowledge of the subject.

Esophagoscopy is considered under the following heads: Anatomy, normal esophagoscopic appearances, technic of esophagoscopy, diseases and anomalies of the esophagus, stenotic diseases of the esophagus, non-stenotic diseases of the esophagus, foreign bodies in the esophagus. Attention is called to the esophagoscopic appearance of these different conditions, whether or not relief is to be obtained by the use of the esophagoscope. The esophagus is thus brought within the sphere of local examination, whatever the conditions be presented, and diagnosis becomes more largely a matter of direct inspection.

Under the head of gastroscopy the author gives a short

history, presenting pictures of the older instruments, such as the polyscope of Trouve and the gastroscope of Mikulicz and Rosenheim. Of these Mikulicz determined that a gastroscope must be rigid, but he gave it a bend, while Rosenheim, who asserted that it should be straight, at times failed to introduce it without a bend. To these points the author adds four more, which he considers requisite for proper gastroscopy:

1. Optic apparatus must be abandoned.
2. The tube must be passed by sight.
3. The stomach must be examined in a collapsed state to permit of mopping, palpation with the instrument, probing and combined endoscopy, and external palpation.
4. General anesthesia is indispensable to prevent retching, during which the diaphragm clamps the tube, rendering exploration impossible.

The technic of gastroscopy is fully described, considerably amplified beyond what was originally given when his paper first appeared in the Fränkel Festschrift number of the ANNALS (December, 1906). It is clear that the author was correct when he stated in this paper that the usefulness of gastroscopy would soon be generally recognized.

A complete summary of the literature (358 references) is given.

The book concludes with five well-made colored plates, showing sixty-eight different views. The whole work is a most creditable exhibition of the rapid and consistent development which has been taking place in laryngology in America during the past twenty years.

A Manual of Diseases of the Nose, Throat and Ear.

BY E. BALDWIN GLEASON, M. D., Clinical Professor of Otology at the Medico-Chirurgical College, Philadelphia. 12 mo. of 556 pages, profusely illustrated. W. B. Saunders Company, Philadelphia and London. Flexible leather. Price, $2.50, net.

This very convenient little volume contains a short, well-expressed treatise on the subject, suitable for students and general practitioners. No attempt is made to be exhaustive, and yet the ground is well covered.

The Labyrinth of Animals.

BY ALBERT A. GRAY, M. D. (Glas.), F. R. S. E. Vol. I. J. S. Churchill, London, 1907. P. Blakiston's Son & Company. $8.40.

The author gives us in this beautiful volume the result of seven years' work upon the labyrinth of vertebrates. The work is of special value to the comparative anatomist and

student of evolution, and the chapter on the significance of anatomic differences in the labyrinths of animals is of unusual interest.

The volume at hand includes the primates, cheiroptera, carnivora, ungulata, edentata and most of the rodentia. The second volume will deal with the rest of the rodents, the insectivora, the cetacea, the sirenia, the marsupialia, the monotremata, as well as the birds, reptiles and amphibia. Fishes have been omitted as having been completely described by Retzius and other anatomists.

For the instruction of the laboratory worker exact directions are given as to the method of making the preparations and of taking the photographs. Briefly, the labyrinth is injected as soon as possible after death, through the oval window with a 5-10 per cent formalin solution. Following this alcohol, ether, colloidin and xyol and paraffin are used during several weeks, and finally the bone surrounding the labyrinth is decalcified and removed and the latter photographed stereoscopically. The photographed labyrinth when viewed through the smell stereoscope which accompanies the book stands out as if made of spun glass.

Some idea of the great value of the book may be had when we remember that the photographs of semicircular canal, vestibule, ampulla, cochlea, etc., are exact representations of the originals, each one in its proper relation to the others and therefore of much greater worth than the opposite pictures made from sections, etc., which adorn the pages of the ordinary anatomical atlas. This is particularly true of the membranous labyrinth. It is also not improbable that the investigation of the pathological changes which cause deafness, tinnitus, etc., made possible by the author's preparation method, may be productive of results of therapeutic value. *Campbell.*

The Principles and Practice of Modern Otology.

By John F. Barnhill, M. D., and Ernest deWolfe Wales, B. S., M. D. 575 pages and 305 illustrations. W. B. Saunders Company, Philadelphia and London.

This is a composite book intended for the use of students and practitioners of general medicine, the anatomy, bacteriology, physiology, and pathology of which is by Dr. Wales and the balance of the book by Dr. Barnhill. The authors have

had in view "To modernize the subject of otology; to correct certain traditional beliefs which have hindered the progress of otology; to advocate the earliest prophylaxis and treatment; to emphasize the importance of a thorough examination and definite diagnosis as a basis for rational treatment and to thoroughly illustrate the subject by suitable pictures."

A point of view similar in the main actuates every author who produces a work of any value. The ideals mentioned have been largely attained and the book here noticed has a right to the title, "Modern Otology."

Dr. Wales has had the hardest though the briefest part of the whole, and if he seems to the reviewer to have failed in any way it is because of the difficulties of the subject and the inability to treat it clearly in the space allowed him. The section on the bony anatomy while rich in pictures is vague in description. A new nomenclature is applied to the semicircular canals, namely, the superior cerebral posterior cerebellar and the tympano-mastoid semicircular canals, a nomenclature much inferior to that now used in Germany, namely, anterior vertical, posterior vertical, and horizontal semicircular canals. The description of the cochlea occupies but thirty lines, and no mention is made of the auditory nerve filaments and their termination. The illustrations are good and really illustrate, but clearness in the descriptive anatomy seems to have been sacrificed in order to save space. Most of the chapter on physiology is a translation from Denker's "Die Otosklerose" and gives the sound wave theories of Helmholz. The bacteriology is described clearly but briefly, and the characteristics of the different bacteria are well analyzed, but the chapter ends with the somewhat startling statement that while the bacteriology of the ear is interesting from a scientific point of view, it is from a practical point of view one of the least important aids in the diagnosis of aural diseases and their complications. With this opinion many aural surgeons will not be in accord.

The chapters written by Dr. Barnhill are clear and concise, and the style is pleasing. The methods of treatment given are those which he has personally found useful, others are for the most part omitted. For the beginner in otology this is excellent and saves much confusion of mind. The illustrations really illustrate the text and are all new. The chapters on

catheterization and on adenectomy are good examples of the clearness of description and of illustration.

In the chapter on acute tubotympanic catarrh the term paracentesis has for the most part been dropped and the term incision of the tympanic membrane been substituted therefor. This change should become universal in aural literature.

The entire subject of mastoid surgery and its complications is most admirably handled, the indications and counter indications for the various procedures being clearly and accurately stated. One point is particularly well brought out in the chapter on the radical operation, namely, the posible danger to the facial nerve when using a protector, on account of the thinness of the upper wall of the Fallopian canal where it forms part of the floor of the aditus ad antrum. The indications and technique for the operation of suturing the facial nerve to the spinal accessory or the hypoglossal is described and a most excellent illustration serves to make it clear.

There is a chapter on the practical relation of suppuration and necrosis to life insurance. The difficult and discouraging subject of non-suppurative otitis media in its various forms is well handled, though there is nothing new in the methods of treatment advocated, those only being given which have stood the test of time. Particular consideration has been given to the things one ought not to do. The surgery of the labyrinth is briefly considered, but detailed methods of diagnosis and treatment are omitted; the subject is doubtless too new for description in a text book. Much more work has yet to be done in this department of aural surgery. There is an excellent chapter on deaf mutism. By a system of cross references much repetition has been avoided and the volume made more useful for reference.

Taken as a whole the volume reflects great credit on its authors and also on American otology, and it can be most heartily commended, not only to the student and general practitioner, but to the aural surgeon as well.

The pictures are remarkably clear for half-tones and show great care in the printing. Many of the illustrations are colored and throughout the book the anatomic plates really illustrate the text and are more than mere pictures.

Richards.

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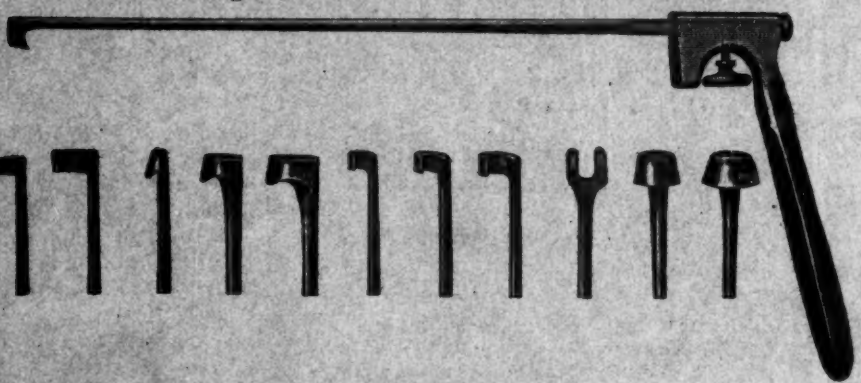

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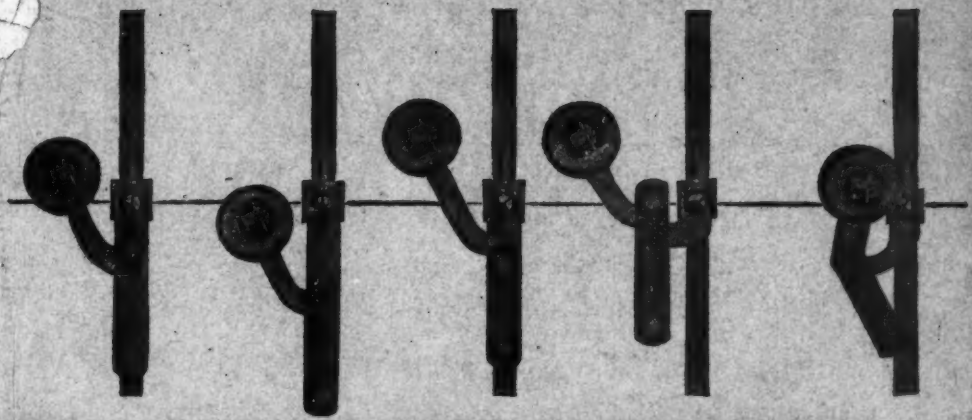

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